CERTIFICATE

This is to certify that this dissertation on **"OUT COME OF SURGERY FOR CHRONIC CALCIFIC PANCREATITIS"** is a bonafide work done by Dr. A. SIVASANKAR under my guidance and supervision, appearing for M.Ch. degree examination branch VI in Surgical Gastroenterology in February 2006.

Prof. R.SURENDRAN, M.S., M.N.A.M.S., M.Ch.
Head of the Department,
Department of Surgical Gastroenterology,
Govt. Stanley Medical College Hospital,
Chennai – 1.

Dr. VASANTHA, M.D.
Dean
Govt. Stanley Medical College
Hospital, Chennai – 1.

Place : Chennai
Date : 28.10.2005

ACKNOWLEDGEMENT

I profusely thank **Dr. Vasantha, M.D.,** Dean, Govt. Stanley Medical Hospital, Chennai, who permitted to pursue research work and to utilize the case material.

I express my sincere gratitude to my chief, **Prof. R.SURENDRAN, M.S., M.N.A.M.S., M.Ch.** for his supreme guidance and supervision, I am extremely thankful to him for his encouragement and kindness throughout the course of my study.

I am grateful to my Assistant professors **Dr. D.Kannan M.S., M.Ch., Dr. T.G.Balachandar M.S., M.Ch., Dr. S. Jeswanth M.S., M.Ch., Dr. S. Rajendran M.S., M.Ch., Dr. P. Ravichandran M.S., M.Ch., and Dr. D. Jyotibasu M.S.,** for preparing the dissertation.

I thank statistician Mr. Venkatesh for analyzing the results.

ABBREVIATIONS

1. LPJ : Longitudinal pancreaticojejunostomy
2. DP : Distal pancreatectomy
3. PJ : Pancreaticojejunostomy
4. LR-LPJ : Local resection of head and Longitudinal pancreaticojejunostomy
5. PG : Pancreaticogastrostomy
6. PD : Pancreaticoduodenectomy
7.. PPPD : Pylorus preserving pancreaticoduodenectomy
8. DPRHP : Duodenum preserving resection of head of pancreas
9. MPD : Main pancreatic duct
10. TCP : Tropical chronic pancreatitis
11. ACP : Alcoholic chronic pancreatitis
12. ERCP : Endoscopic retrograde cholangiopancreatography
13. MRCP : Magnetic resonance cholangiopancreatography
14. ESWL : Extracorporeal shock wave lithotripsy
15. PA : Pseudoaneurysm
16. OHA : Oral hypoglycemic agents
17. UTI : Urinary tract infection
18. GI : Gastrointestinal
19. OR : Odds ratio
20. CI : Confidence interval
21. CP : Chronic pancreatitis
22. CRISP3 : Cysteine-rich secretory protein-3

CONTENTS

DECLARATION

I, **Dr. A. SIVASANKAR,** solemnly declare that the dissertation titled **"OUT COME OF SURGERY FOR CHRONIC CALCIFIC PANCREATITIS"** was done by me at Government Stanley Medical College Hospital, Chennai – 1 during the academic year 2003 – 2006 under the guidance and supervision of my chief, **Prof. R.SURENDRAN, M.N.A.M.S., M.Ch.**

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai towards the partial fulfillment of requirement for the award of M.Ch. degree branch VI in Surgical Gastroenterology.

Place : Chennai **Dr. A. SIVASANKAR**

Date : 28.10.2005

INTRODUCTION

The management of patients with chronic pancreatitis remains a difficult and challenging problem. The most common cause of chronic pancreatitis in western countries is chronic alcohol abuse, opposed to the tropical chronic pancreatitis, commonest cause in tropical countries. Tropical chronic pancreatitis is a unique form of chronic nonalcoholic pancreatitis, which is limited to the tropical developing countries. Patients present at a young age with severe abdominal pain, weight loss and insulin requiring diabetes.

Recently, gene mutations, which lead to hereditary and idiopathic chronic pancreatitis, have also been reported 1. Patients come to the doctor with clinical symptoms such as maldigestion, severe weight loss, recurrent severe upper abdominal pain and therefore, a greatly impaired quality of life. Pain is a predominant symptom in chronic pancreatitis. Later in course of the disease, exocrine and endocrine insufficiency may develop. The pain of chronic pancreatitis patients may prove difficult to control, and addiction to analgesics-opioids is not uncommon.

The therapy for chronic pancreatitis consists primarily of conservative and symptom related treatment but long-term follow-up studies have demonstrated that about 50% of the patients will undergo surgical treatment at sometime in the course of the disease 1.

Before 1980, pancreatic surgery was associated with an operative mortality rate of up to 25% and the results of surgery were unpredictable, with many patients continuing to suffer from severe pain often associated with marked endocrine and exocrine insufficiency 2. Although conservative treatment may be successful in some patients with chronic pancreatitis if spontaneous remission of pain occurs, its timing is unpredictable and the patient may lose valuable years of life 2.

The principal indication for surgery in chronic pancreatitis is intractable pain, which prevents the patient from leading a normal life. Less frequently, surgery is indicated for the treatment of complications of chronic pancreatitis including pseudocyst, pancreatic fistulae, splenic vein thrombosis, bleeding pseudoaneurysms, and duodenal or biliary obstruction. Additionally the

exclusion of pancreatic cancer is sometimes not possible even with all the diagnostic procedures available, so that only surgical resection can bring a definite answer.

Controversy still exists regarding the optimal timing of surgery in these patients, but by the time they are referred to the surgeon, patients are often in a state of opiate and alcoholic addiction, undernutrition and sometimes diabetes or exocrine insufficiency 3.

Recently some studies have shown that surgery has a positive influence on the course of the disease, postponing the final "burnout" of the pancreas and the consequent appearance of exocrine and endocrine insufficiency 1. Therefore, it is of great clinical importance in the treatment of chronic pancreatitis to address the above mentioned problems surgically before the disease has progressed to an advanced stage in which the exocrine and endocrine function is completely lost.

The ideal operation should provide the best long-term results with minimal functional impairment. However, the optimal surgical technique has never been established. Even among specialist the choice between pancreatic resection and a more conservative approach is controversial. Two main types of operation have been developed based on different theories of pain pathogenesis. The drainage approach is based on the hypothesis of ductal and/or parenchymal hypertension. The resection approach is based on the hypothesis of local inflammation and perineuritis. Drainage procedures include Puestow procedure, longitudinal pancreaticojejunostmy, pancreaticogastrostomy and local resection and longitudinal pancreaticojejunostomy (LR-LPJ). Resectional procedures include distal pancreatectomy, central pancreatectomy, Duodenum preserving pancreatic head resection (Beger procedure) and proximal pancreatectomy - Whipple procedure or pylorus-preserving pancreaticoduodenectomy. Operations may be associated with a better long-term outcome and with a minimal mortality rate in specialist units. Several centers report an operative mortality rate below 5 percent for pancreaticoduodenectomy, including a number of institutions with no perioperative deaths in large series of patients. Postoperative morbidity, however, remains high after these procedures although most complications are amenable to treatment.

Operations can be tailored to the particular pattern of disease in an individual patient, but clear guidelines are lacking. Moreover, the impact of surgical intervention on the natural history is not yet well understood. Theoretically, improvement of

pancreatic function may occur after drainage procedure. After resection, pancreatic function is known to deteriorate 1.Given that chronic pancreatitis is basically a progressive lesion of pancreatic parenchyma, and that a significant part of this group of patients represents a sector of people living on the peripheries of society, who consume heavy amounts of alcohol or addict to narcotics and lead unfortunately self-destructive lives, the initial promising surgical results have usually deteriorated on a long-term basis. The late mortality rate is high, ranging between 20-40% and is similar to that of some malignant disease 6. Due to these factors and the poor compliance of these patients, results covering long-term follow-up period are few and far between literature.

REVIEW OF LITERATURE

The main purpose of operative management of chronic pancreatitis is to relieve symptorns and to treat complications of the disease. The main indication for elective operation is to alleviate pain, the pathophysiology of which is not clearly understood. Some studies, however, have associated pain with increased ductal pressure4, which is the result of obstruction either by stone or by stricture. This explains why duct decompression has been successful in relieving chronic pain.

Recently, it has been shown that resection of head of pancreas (pacemaker of the disease [5] which harbors ductal and / or parenchymal pathologies in 95 % of patients with chronic pancreatitis) effectively relieves pain. Realizing this, the surgeon is faced with the decision to choose an optimal procedure for the patient. It should provide pain control and quality of life while being safe and free of side effects.

NATURAL HISTORY:

The natural history of pain in patients with chronic pancreatitis has been evaluated primarily by retrospective observational studies 6, 7. These studies have included patients with heterogeneous etiologies of pancreatitis and a wide array of potential manifestations of disease. Although insight can be derived from them, care should be used when drawing firm conclusions.

Most of these studies have suggested that the pain associated with chronic pancreatitis diminishes with time. There is a debate; however, regarding the time it takes for pain to abate, whether this occurs in a majority of patients with uncomplicated disease, and whether pain cessation inversely parallels exocrine pancreatic dysfunction. Ammann et al 8 in a study of predominantly alcohol-induced pancreatitis found that 85% of these patients had cessation of pain after a median of 4.5 years, and this pain relief was not altered by surgical intervention. This study also found that pain relief was associated with deterioration of pancreatic function and the onset of pancreatic calcification. There was a wide range of time to pain cessation, however, among patients whose pain eventually abated (0 to 18 years). Also, 50% to 60% of patients had their last episode of pain despite the presence of pancreatic calcifications. The majority of patients who had pain despite having pancreatic calcification had pancreatic pseudocysts. In a second study, Lankisch et al 6 found that after a median follow-up of 11.3 years, significantly fewer patients had painful episodes, but the majority of patients still experienced pain. Similarly, the overall numbers of patients experiencing pain relief were significantly greater if patients developed pancreatic exocrine insufficiency or pancreatic calcifications. The majority of patients who had exocrine insufficiency or pancreatic calcification, however, continued to experience pain. Interestingly, pain relief could also not be predicted by the presence of pancreatic duct abnormalities. This study also showed that there was no difference in the long-term pain course between patients who were operated on and those who were not. In the short-term, 89% of patients who underwent surgery experienced relief of pain. The comparison of patients who underwent surgery and those who did not should be taken with caution in both of these studies because these groups may not have been comparable, and the surgical procedures used in the majority of patients are not necessarily standard presently. These studies also mixed patients undergoing surgery for uncomplicated and complicated chronic pancreatitis (i.e., pseudocysts). A third study by Layer et al 7 also found that the frequency and severity of pain decreased with time, but that this decrease in pain was independent of exocrine insufficiency or pancreatic calcification. The etiology of the chronic pancreatitis affected the course of pain in this study. Patients with early onset idiopathic chronic pancreatitis were more likely to have severe pain as an initial symptom than patients with late-onset idiopathic or alcohol-induced chronic pancreatitis.

In general, although pain may decrease over time in patients with chronic pancreatitis, this should not be relied upon, especially in patients with severe discomfort. Medical and interventional therapies designed to diminish and expedite pain relief

should be thoroughly investigated. Although development of exocrine insufficiency and pancreatic calcification may be associated with pain relief, there is no clear evidence that this association is causal.

PANCREATIC DUCT DECOMPRESSION PROCEDURES:

The assumption that ductal ectasia, secondary to obstruction or stenosis of the pancreatic ductal system is indicative of intraductal hypertension constituted the basis for pancreatic ductal decompression at the beginning of the last century.

Jalleh et al, in a study to determine the contribution of parenchymal hypertension to pain, pancreatic tissue pressures were measured intraoperatively in patients with chronic pancreatitis undergoing pancreatic surgery, it was found that the pressure (mean ± s.e.m) was substantially elevated in all regions of the pancreas compared with reference subjects: head (257 ± 59 versus 19 ± 5 mm Hg, $p < 0.05$), body (201 ± 51 versus 13 ± 6 mm Hg, $p < 0.05$) and tail (161 ± 45 versus 11 ± 3 mm Hg, $p < 0.05$). Elevation was greater in areas of calcific disease (281-383 mm Hg) than in non-calcific disease (81-120 mm Hg, $p < 0.05$). The greatly increased tissue pressures in chronic pancreatitis, especially in the presence of calcification, suggest a possible 'compartment syndrome'.[9]

After the original reports by Du Val and Zollinger, the technique for surgical decompression of the pancreatic ductal system in patients with chronic pancreatitis was modified first by Puestow and Gillespy and later to the longitudinal pancreaticojejunostorny proposed by Partington and Rochelle. Because the low surgical morbidity and mortality of pancreatic duct internal drainage and the good early results achieved, primary ductal decompression became the gold standard treatment of pain in patients with chronic pancreatitis, presenting with a dilated pancreatic duct.

Longitudinal pancreatico jejunostomy (LPJ) is safe. Many authors have reported low mortality rate (0 %) [10,11]. Although longitudinal pancreaticojejunostomy has not been evaluated in controlled randomized trails, a technical review from the AGA on treatment of pain in chronic pancreatitis concluded 'current consensus among surgeons is that drainage operations are safest and most likely to be effective' [12]. Some studies suggest that pain control achieved by pancreatic decompression is higher in nonalcoholic pancreatitis than in alcoholic patients [13]. In contrast, another report failed to find any difference in the pain relief provided by lateral pancreaticojejunostomy between alcoholic and abstinent patients [14].

The main advantage of lateral pancreaticojejunostorny is the maintenance of pancreatic exocrine and endocrine function. In the past, the demonstration of a small but significant loss of pancreatic exocrine and endocrine function after pancreatic drainages procedures was mostly obtained from retrospective, nonstandardized studies [15]. Two prospective, randomized studies have shown that successful pancreatic duct internal drainages may actually postpone the appearance of exocrine and endocrine insufficiency[16, 17]. The hypothesis that neural inflammation is an important pathologic mechanism of pain and the high incidence of ductal alteration and\or inflammatory mass in the head of pancreas motivated the concept of resection of inflammatory tissue as the treatment of choice of pain in chronic pancreatitis[18.] Pathological studies in combination with modern molecular biology investigations, suggest that the pancreatic head is the "pacemaker" of the diseases in most of patients with chronic pancreatitis [19]. However the triggering mechanisms for pancreatic enlargement are still not known, in particular it is unclear what trigger growth factors to step into action.

RESECTIONAL PROCEDURES:

For the past 15 years pancreaticoduodenectomy has been the procedure of choice in many institutions for treating patients with chronic pancreatitis presenting with pancreatic head complications. Twenty years ago, the reported operative mortality for pancreaticoduodenectomy was unacceptable high in many centres (about 20%). With increasing experience with proximal pancreatectomy for cancer, operative mortality has progressively decreased so that nowadays most centres that treat large numbers of patients have an operative mortality well below 5%. Indeed, operative mortality of less than 2% is reported with increasing frequency in series of more than 50 patients[20, 21]. Although postoperative morbidity approaches 40-50%, complications are largely manageable and reversible. A positive loop has developed in which the reduction in mortality and morbidity has led to wider applications of proximal pancreatectomy, which in turn increases surgical experience and further improves results[22]. Pancreaticoduodenectomy for chronic pancreatitis also has an obligate potential long-term morbidity. In patients with a normal pancreas, PD has no important clinical effect on endocrine or exocrine reserve. However, in the context of chronic pancreatitis, pancreatic endocrine and exocrine insufficiency often develops in the absence of any surgical resection as the natural course of the diseases. Because proximal pancreatectomy removes about 40% to 60% of the pancreatic parenchyma, the possibility of prematurely precipitating postoperative pancreatic insufficiency must be considered when such an approach is contemplated. Although exocrine insufficiency had developed in an

additional 26% of patients at follow-up and endocrine insufficiency in an additional 30%, the mean time to onset of diabetes mellitus was 4.7 years postoperatively. While this suggests that the chronic fibrosis and glandular destruction of chronic pancreatitis progressed in the pancreatic remnant, the proximal parenchymal resection possibly contributed to a more premature loss of pancreatic endocrine and exocrine reserve and disruption of enteroinsular axis. Similar long-term sequelae of the combination of chronic pancreatitis and proximal pancreatectomy have been well described previously [23].

Distal pancreatectomy is a safe procedure (perioperative mortality 0-3.8%, morbidity 15-31%) that may be performed with or without splenectomy [24, 25]. It provides long-term pain relief less reliably than PPPD or Classical PD unless the pancreatic inflammatory process is specifically localized to the body or tail of the gland (eg. obstructive pancreatitis secondary to a ductal stricture). Even in patients with such focal distal disease, pain relief with distal pancreatectomy has been inconsistent [25, 26, 27]. Rattner et al. reported good pain relief in only 31% of patients undergoing distal pancreatectomy for distal chronic pancreatitis uncomplicated by a pseudocyst [26]. Sawyer and Frey, on the other hand, found adequate pain relief in 90% of those with distal disease at a mean follow-up of 4 years [27]. They emphasized that distal pancreatectomy for chronic pancreatitis should be utilized only in appropriate patients (duct < 5 mm diameter, disease seen on CT and ERCP to be restricted to the pancreatic body, tail or both), a population comprising 5-15% of patients with chronic pancreatitis [27]. In a more recent review of 40 patients with a single, prominent stricture of the main pancreatic duct and chronic pancreatitis in the obstructed segment, Sakorafas et al. found that distal pancreatectomy produced significant pain relief in 81% of patients at a mean 6.7 years of

follow-up [25]. Eventually, 45% and 47% of these patients developed postoperative endocrine and exocrine insufficiency, respectively [25].

Distal left to right sided resections (60-90% distal pancreatectomies) and total pancreatectomies present considerable morbidity and mortality rates related to the metabolic consequences of massive resection [28]. Induction of endocrine and exocrine insufficiency even after distal pancreatectomy leads to multiple problems with nutritional management.

COMBINED RESECTION AND DRAINAGE PROCEDURES:

The association of pancreatic head resection with a longitudinal pancreaticojejunostomy, combining with advantages of maximization of ductal decompression provided by the drainage procedure and the efficacy of cephalic pancreatic resection in pain control, has been used with good results. Beger et al were the pioneers of the duodenum-preserving pancreatic head resection. The innovative subtotal head resection involves an anatomic resection of part of the head and uncinate process beginning just to left of intrapancreatic portion of common bile duct and extending to the neck of the gland. This leaves a minute portion of pancreatic tissue along the medial wall of the duodenum and all the body and tail of the gland. Internal drainage is restored by two pancreaticojejunostomies, one to the body/tail region, with addition of a longitudinal pancreaticojejunostomy if necessary, and a separate anastomosis to the paraduodenal pancreatic remnant. The rationale of this procedure is to remove only part

of the diseased pancreatic head, preserving the extrapancreatic organs including the duodenum with its role in regulation of glucose metabolism.

The most commonly used modification of the duodenum-preserving pancreatic head resection was originally proposed by Frey and Smith in 1987 in which a core of pancreatic head is resected nonanatomically, leaving a rim of pancreatic tissue adjacent to the portal and mesenteric veins. This limited resection of the head of the pancreas is technically easier to perform than the Beger operation because it does not necessitate transaction of the gland above the portal vein and thereby minimizes the risk of a bleeding complication, especially in the presence of extrahepatic portal hypertension. In chronic pancreatitis, the crucial triangle lies between the distal common bile duct, the wirsunigian duct and the superior mesenteric-portal vein. This region is addressed by classical resection of PD, by PPPD, by DPRHP and by Frey procedure.

The overall results of these DPRHP and LRLPJ show long-term relief in 75-95% of patients [29, 30]. Two prospective, randomized studies conducted by the same group in two different periods, compared the DPRHP and LRLPJ. In both trials, the two procedures were equally effective concerning pain relief, preserving of pancreatic function and quality of life [31, 32]. The postoperative morbidity, however, was significantly lower with LRLPJ. Two other prospective, randomized trials comparing the DPRHP either to PPPD or to the classical PD [5, 33] showed that pancreatic exocrine and endocrine function was preserved by the Beger procedure, which was also superior to PD regarding long-term pain relief in one trial [5]

Avoiding late postoperative complications must be a matter of concern in surgical treatment of chronic pancreatitis. Considering the most imperative issue of

sustained quality of life improvement, the different variations of duodenum-preserving pancreatic head resections proved superior in comparison to PD, including the pylorus-preserving modality. The excellent results obtained with these methods underlie that organ-sparing techniques should be preferred in the surgical treatment of chronic pancreatitis.

There are several reasons why duct decompression might fail to control pain in patients with chronic pancreatitis, as detailed in a report by Markowitz et al [34]. These include pancreaticojejunostomy anastomosis that is inadequate or stricture. Strictures may also affect the intrapancreatic segment of the common bile duct and the duodenum, and are particularly likely to do so in the large duct form of chronic pancreatitis. Recurrent or persisting acute inflammation in the gland can produce pain, pseudocyst or even abscess. Pancreatic cancer was unsuspected but present in two of the patients in our series. Markowitz reported 13% of unrecognized pancreatic cancer in their series [34].

Early failures, may have been because the wrong operative strategy was chosen initially. Late failures may have been the result of disease progression, neuritis or other factors. Continued ethanol consumption has been associated with the greater likelihood of continuing or increasing pain [35].

Strategies for remedy of pain are dependent of the investigative workup. Redrainage of the pancreatic duct, limited resection of distal undrained segments and biliary decompression can benefit selected patients. The options for those patients are limited. Because of the anatomic evidence for a sensory neuropathy in the head of pancreas in chronic pancreatitis [36] and because a Whipple-type or duodenum-sparing resection of the pancreatic head have been reasonably successful as primary surgical

therapy for chronic pancreatitis, Markowitz et al have employed pancreatic head resection in an attempt to salvage a poor outcome after pancreaticojejunostomy in a 73% of patients (11 patients of 15). They obtained 40% pain-relief and 30% partial relief in addition. Two patients were subjected for completion pancreatectomy [34]. Whole pancreas and islet cells transplantation [37] are under investigations as alternative adjuncts to this problem.

ALCOHOL ABSTINENCE:

Most 35, 38 but not all 6, retrospective studies suggested that alcohol abstinence leads to reduction of pain in individuals with chronic pancreatitis. There have been no prospective studies performed, however, to ascertain directly the results of alcohol abstinence on reduction of pain in these patients. Strum 38 reported the clinical experience regarding abstinence and long-term outcome in patients with alcoholic chronic pancreatitis. Forty-two patients had an average 5.4 years of follow-up. Disability or death occurred in all patients who continued to drink heavily as compared with 38% of those who abstained or diminished their intake. A pooled analysis was also performed, including seven case series, comprising over 450 patients. Abdominal pain was significantly less common in those who stopped alcohol intake. Lankisch et al did not find a significant difference in pain relief between patients with alcohol-induced chronic pancreatitis who stopped drinking and those who did not 6. There is also some evidence that patients who continue to drink are more likely to develop pancreatic complications 39. Brinton et al 40 reported results of lateral pancreaticojejunostomy in 39 patients with chronic pancreatitis and found no correlation between the pain relief in alcoholic chronic pancreatitis and those who were not alcoholic . This is in contrast to report by Sato et al, who found that over mean period of 9.1 years only 50% of patients with alcoholic pancreatitis had a good result after surgery compared to 83% of those who had nonalcoholic pancreatitis 41. Another report by Sharma et al 42 found that 88% of patients of patients with nonalcoholic chronic pancreatitis had immediate and lasting pain relief.

In the only major report on the results of surgery in 33 patients with nonalcoholic chronic calcific pancreatitis from South India, 77% of 26 surviving patients had good relief of pain at a median follow-up of 28 months 43.

ENDOCRINE AND EXOCRINE INSUFFICIENCY:

Diabetes occurs in 85% of patients with chronic pancreatitis over a 25-years period. Nearly 50% of these patients need insulin therapy. In a 3-yrs. study on 70 patients with tropical calcific pancreatitis, (75 % had pain, 52% diabetes, 28 % weight loss, 2 % steatorrhea). 22 patients underwent LPJ 44 , most insulin requiring diabetic patients achieved a reduction in insulin dosage or could stop insulin altogether. In an another study 45 LPJ was performed in 10 patients with tropical chronic pancreatitis (diabetes in all, pain in 6, steatorrhea in 6). At 1-year follow-up, abdominal pain was relieved with patients experiencing weight gain, improved working capacity and quality of life. Their mean weight increased from 34 to 38.3 kg. Of 6 patients with steatorrhea, three were fully relieved, 3 had partial relief and, one still had steatorrhea and yet had gained 11 kg of body weight over 1-1/2 yr. The mean insulin requirement fell from 46 U to 34 U per day. Nealon et al 46 reported that in patients with mild moderate chronic pancreatitis, operative decompression of pancreatic duct early in the course of the disease did not improve but halted the progressive loss of endocrine and exocrine functions. Prospective evaluation in 87 patients, who underwent decompression of duct to relieve abdominal pain, was reported. Among them, 17 patients were readmitted to operative or nonoperative treatment. Nine of these randomized patients were operated upon and eight were not. Seven of nine (78 %) patients retained their functional status in follow-up, whereas only two of eight patients (25%) randomized to nonoperation preserved their functional status. These authors supported the concept that high intra – ductal pressure may contribute to the ongoing loss of function and perhaps to an ongoing level of subacute inflammation, which restricts utilization of nutritional substrates.

Hammel et al 47 who, over a follow-up period of 15 year found that the prevalence of diabetes was 21% in 222 patients who had undergone surgery, in contrast to 33% in 224 patients managed conservatively. Similar findings were reported 48 after Extra

corporeal Shock wave Lithotripsy (ESWL) of pancreatic stones when 23 (77%) out of 30 patients with steatorrhea improved, as did two of five patients with abnormal glucose tolerance.

In another study, Brand et al 49 reported pain relief in 81.6%, an improved quality of life in 68.4% and weight gain in 68.2% (with prior weight loss) of patients after endoscopic drainage of the pancreatic duct with ESWL of pancreatic stones in 48 patients with chronic calcific pancreatitis over a period of 1 year. Pancreatic steatorrhea improved in 10/13 (67.9%) patients, and normalization of endocrine pancreatic function occurred in five patients ($p = 0.45$); median glycosylated hemoglobin (Hb A1C) levels decreased significantly after pancreatic ductal drainage.

In an Indian study involving 53 patients with tropical chronic pancreatitis who underwent modified Puestow procedure, prospective comparison was done between 46 operated patients who completed 5 year of follow up with 40 patient who did not undergo operation 50 41/46 operated patients (89%) had complete pain relief, whereas in those managed conservatively, even though they had milder symptoms, Pain relief occurred in only 58%. In the operated group, the mean fasting (209 mg%) and postprandial (320 mg%) blood glucose and insulin requirements (40 units/day) decreased postoperatively (fasting 162 mg%, Postprandial 254%. Insulin requirement 18.2 units/day; $p < 0.01$), and steatorrhea improved in one of six patients. In the nonoperated group, endocrine and exocrine pancreatic function remained unchanged. Authors have shown that patients with tropical pancreatitis who undergo the modified Puestow's procedure not only have relief from pain but also improvement of diabetes.

In an another Indian prospective study of pancreatic β cell and exocrine function following duct decompression in tropical calcific pancreatitis by E. Bhatia et al 51 , reported that although there was a significant improvement in the pain score with a complete remission of pain in > 90% of patients, there was no change in β- cell function and exocrine function after 1 year of follow-up.

In alcoholic chronic pancreatitis, the results of decompression surgery have been inconsistent with regard to improvement in endocrine and exocrine functions. Adams et al 11 in a retrospective study of 85 patients with alcoholic chronic pancreatitis, showed that insulin use continued in 23% and that taking of pancreatic enzyme supplements persisted in 34% after the modified Puestow's procedure. The author suggested that pancreatic exocrine and endocrine functions worsened after drainage surgery.

In Amman's study 8 drainage surgery did not delay the development of exocrine or endocrine insufficiency in chronic alcoholic pancreatitis patients. The inconsistency of results in alcoholic chronic pancreatitis may reflect the difference in the extent of pancreatic damage at the time of surgery and whether or not the patient has stopped drinking.

Ramesh and Augustine 52 reported that glucose tolerance improved in only 18% of their TCP patients, while Sharma et al 13 noted that these was no significant change in insulin requirement after intervention.

The two most striking changes in tropical calcification pancreatitis are marked atrophy of exocrine pancreas but more involving the islets of Langerhans instead the latter shown on consistent hypertrophy and hyperplasia. Further there is an unequivocal evidence of nesidioblastosis in tropical chronic pancreatitis.

Nesidioblastosis is a multifocal, ductuloinsular proliferation involving all cellular components of the islets and is usually seen in hyperinsulinemic hypoglycemia of childhood. Histochemical staining using immunoperoxidase has shown a strong activity for insulin in both existing and newly formed islets. Histochemical evidence thus suggests continued insulin production. The insulin changes in tropical chronic pancreatitis are therefore paradoxical. Despite insulin hyperplasia and hypertrophy, the patient suffers from a resistant form of diabetes mellitus. The role of islets in the etiopathogenesis of the brittle, but insulin-dependent diabetes is very intriguing. One mechanism may be failure of release of insulin from islets. The other may be presence of circulating antibodies, resulting in brittle diabetes with requirement of very high doses of insulin and frequent episodes of hypoglycemia.

The most striking findings in alcoholic chronic pancreatitis, on the other hand are ductal dilatation with protein plugs, fat necrosis, parenchymal necrosis, acute inflammatory reaction, and parenchymal calcification 50. The ductoinsular changes such as islet hypotrophy/ hyperplasia and nesidioblastosis, characteristically seen in tropical chronic pancreatitis, as mentioned earlier are rarely seen in alcoholic pancreatitis. This difference is pathology may explain the variable changes in pancreatic dysfunction after drainage surgery in alcoholic chronic pancreatitis as opposed to that in tropical chronic pancreatitis.

The possible explanation for the improvement in endocrine function after decompressive surgery in tropical chronic pancreatitis may lie in the fact that in this disease, the islet cells may be normal or even increased in number 50 , in fact the K value (the slope of disappearance of glucose from the blood after intravenous injection) may be normal in patients with tropical chronic

pancreatitis who have overt pancreatic diabetes. It is postulated that obstruction of the pancreatic duct, together with the dense intralobular and perilobular fibrosis, creates a compartment syndrome and that the insulin deficiency results from poor islet perfusion syndrome and the poor insulin absorption due to raised interstitial pressures. There is experimental evidence 53 to prove that ductal decompression and surgical incision of the gland improve the blood flow to the pancreas and lower the increased interstitial and perfusion pressures. The improvement of pancreatic ischemia may be associated with relief of pain, and the enhanced perfusion of the islet of Langerhans may increase insulin transfer and absorption, thereby resulting in an improvement of the disease.

The principal criterion to determine candidacy for ductal drainage in Nealon's series was the presence of a dilated pancreatic duct. Thus, what the study actually reports is the outcome of pancreatic drainage in patients with dilated ducts versus the natural history of patients with chronic pancreatitis and no ductal dilatation. It is therefore uncertain whether the slow deterioration of pancreatic endocrine function in patients who underwent surgery is actually caused by surgical drainage procedures.

Distal pancreatectomy was associated with increased risk of diabetes mellitus which has been suggested by several reports [25] . However, most of these studies followed up small number of patients only, often for a short time and did not compare distal pancreatectomy with other elective pancreatic surgical procedures, or lacked a control group. In a review of seven studies including a total of 100 patients with chronic pancreatitis followed up for 3.6 to 7.0 years after a 40 to 80% distal pancreatectomy have shown that the prevalence of IDDM increased from 0 to 10% preoperatively to 19%-48% in the late postoperative period [53] . Increasing the extent of distal pancreatectomy to 80 to 95% led to a postoperative rate of diabetes mellitus of 58% to 100% and increased late mortality [25,26] .

The influence of pancreaticoduodenectomy on the risks of postoperative diabetes mellitus is unclear 54, 55. It has been shown that pancreaticoduodenectomy results in decreased insulin secretion after surgery 56. In a review of eight series including a

total of 325 patients with chronic pancreatitis followed up between 25 months and 6.2 years after pancreaticoduodenectomy found an increase in the rate of IDDM from 0% to 16% preoperatively to 11% to 68% in the late postoperative follow-up 53.

Following conventional pancreaticoduodenectomy or PPPD, patients showed about a 20% decrease in the PABA test or decreased faecal chymotrypsin 57. In this group, insufficient weight gain despite intake of regular amounts of pancreatin was shown to be associated with decreased pancreatic exocrine function postoperatively 58 .

In patients with preexisting chronic pancreatitis, the percentage of patients with steatorrhoea was increased from 3.7% to 19% by 40- 80% distal resection, from 9.0% to 37.6% by 80-95% distal resection and from 5.2 to 55% by Whipple procedure. The average faecal loss of fat following a 80-95% distal resection in patients ingesting 100g fat per day was 26.4 g/day (normal: less than 5-7 g/d), that is, the coefficient of fat absorption in these patients was reduced to 73.6% compared with greater than 93-95% in healthy humans 59. Residual lipid digestion in spite of almost complete loss of pancreatic exocrine tissue is probably due to gastric lipase.

ASSOCIATED COMPLICATIONS:

Pseudo cysts complicate chronic pancreatitis in 20-40% of patients and unlike those secondary to acute pancreatitis, tend to persist and cause further complication [61]. In a report from Nealon et al [60], duct drainage alone in a chronic pancreatitis (dilated duct), patients with pseudo cyst is sufficient and effective in completely resolving associated pseudocysts. They have shown that dynamics of the ductal system completely determine the course of pseudocysts in patients with chronic pancreatitis. They placed intra operative external drains in the pseudocysts of 33 patients who had LPJ alone, and the output of these drains was essentially zero immediately after operation. The fact that external drain outputs dropped so promptly further illustrates the dominant role that ductal pressure play in pseudocyst dynamics.

There are reports of conservative treatment of pancreatic pseudocysts [61,62], but the majority of pseudo cysts, especially when symptomatic, require surgical or interventional approach [63] . The major modalities of therapy include endoscopic or percutaneous drainage and surgery for internal drainage or resection. ERCP use can predict treatment success and, in our study, rendered a protective mortality effect [64]. Accumulating evidence suggests that more emphasis should be given to ductal anatomy than cyst characteristics. Studies have confirmed that main pancreatic duct complete obstruction is a poor prognostic factor of percutaneous drainage [64,65]. The benefit of ERCP may be both diagnostic and therapeutic given the ability to endoscopically stent the main pancreatic duct. Caution should be exercised in stenting chronic pancreatitis given the strong potential for numerous stent exchanges over the long course of the disease. ERCP use likely should be increased given its benefit and low use, as shown by others [64, 66] . Increased use of ERCP should be tempered by the potential adverse events associated with ERCP use, particularly at an institutional or a provider level. In addition, the advent of MRCP may address of the pancreatic duct while avoiding some of the adverse events associated with ERCP. Endoscopic therapy, although successful in some series [67] , has limitations and external drainage often leads to a persistent pancreatic fistulae. Last, surgical drainage of pseudocyst is safe, effective, and enduring [64,66]. Clear indications for surgical drainage remain large or multiple cysts; cysts with adjacent organ involvement, particularly splenic involvement; biliary pancreatitis and chronic pancreatitis.

Surgical internal drainage can be preformed either to the stomach or to a jejunal limb. In patients with pseudocysts and main duct dilatation, internal drainage of the cyst

must be associated to a longitudinal pancreaticojejunostomy. The good results obtained with this technique stress the importance of providing an outflow route for the obstructed pancreatic duct, and not just for the pseudocyst. Pancreatic pseudocysts complicated by splenic parenchymal involvement respond poorly to percutaneous drainage and are preferably treated by distal pancreatectomy and splenectomy. Considering the diversity of their presentations, the management of pseudocysts should be tailored to meet each patient's individual needs. As there have been no prospective, randomized trails comparing head to head result of the three available forms of therapy further studies are needed before percutaneous drainage or endoscopic approach can be definitely recommended as the preferred initial mode of pseudocyst management. Potentially, percutaneous drainage could be used as a "bridge"procedure for patients who have an infected pseudocyst, are malnourished, or are poor surgical risk. Clear end points for failure of percutaneous drainage are necessary. Endoscopic drainage may have specific clinical application in pseudocysts in the head of the pancreas or small persistent pseudocysts.

In a review, terminal biliary stenosis complication reportedly occurs in 3 % to 23 % of patients with chronic pancreatitis [68]. Preoperative diagnosis was based on history of jaundice, marked elevation of alkaline phosphatase, or both and visualization of the lesion on endoscopic retrograde studies. Failure to identify and correct this complication may result in continued disability including pain, recurrent cholangitis, and ultimately biliary cirrhosis.

Determining whether biliary obstruction is due to chronic pancreatitis or malignancy can be a very challenging clinical problem. This differentiation is of utmost

importance since malignancy will require resection whereas a biliary drainage procedure would suffice for benign disease. Wapnick and associates have defines a number of parameters, which make a diagnosis of inflammatory disease of the pancreas more likely than cancer [69] . In their study, patients with chronic pancreatitis were significantly younger than patients with carcinoma, having an average age of 47 versus 62. Total serum bilirubin was much higher with pancreatic malignancy being 18.5 mg/dl compared with 5.6 mg/dl in chronic pancreatitis. Perhaps more important than the absolute rise of bilirubin was the pattern of elevation. In patients with malignancy, bilirubin rose progressively until the biliary tree was decompressed, while with chronic pancreatitis, bilirubin rose to an apex and then fell as the attack resolved. In other words, jaundice usually waxes and wanes in chronic pancreatitis while in cancer it increases relentlessly [69]. All patients with pancreatitis in their study had an admission bilirubin that fell below 5 mg/dl within two weeks; in all patients with malignancy the level continued to rise. They concluded that the course of the bilirubin during the first seven to ten days of hospitalization was the single most accurate test distinguishing carcinoma from pancreatitis. Likewise, Frey and coworkers found that serum bilirubin was seldom higher than 10 mg/dl in chronic pancreatitis and usually diminished within 7-10 days as the inflammatory exacerbation subsides [70]. Clinically, patients with carcinoma usually have a brief antecedent history of abdominal discomfort with an unremitting elevation of bilirubin. Patients with chronic pancreatitis have symptoms of recurrent upper abdominal and back pain that has been present for some time prior to the development of jaundice. Radiological studies can suggest malignancy but are not absolutely diagnostic. A fixed high-grade, short stenosis or complete obstruction of intrapancreatic bile duct most often

suggests carcinoma the head of the pancreas, distal bile duct or ampulla of Vater. In pancreatic carcinoma, a “shelf” from the tumour is usually seen with cholangiography. The gradual tapering of the intrahepatic duct in chronic pancreatitis should distinguish it from the abrupt, short obstruction seen in pancreatic cancer. However, exceptions to this rule are sufficiently frequent to render cholangiographic findings unreliable in distinguishing these two disease processes.

Both pancreatic cancer and chronic pancreatitis rarely appear as duodenal obstruction without causing jaundice. Patients with carcinoma of the pancreas who have duodenal obstruction without jaundice are usually found to have widespread metastatic disease at the time of surgical exploration. An irregular stenotic lesion in the duodenal sweep is highly suggestive of malignancy, particularly when associated with obstructive jaundice. Radiologically, the appearance of pancreatic malignancy is that of a circumferential constriction suggesting mucosal invasion, or fixed, distorted folds with nodularity, and speculation . Endoscopically, reactive duodenal changes including marked mucosal friability and nodularity are seen with both conditions. In summary, no single clinical, biochemical, or radiological feature reliably differentiates a biliary stricture or duodenal obstruction secondary to chronic pancreatitis from one due to pancreatic cancer, A high degree of clinical suspicion is needed to be certain one is not missing a pancreatic cancer.

The alternative choices for the management of biliary stricture this complication are Choledochoduodenostomy and Choledochojejunostomy. Cholecystojejunostomy may be the only surgical option for surgical biliary decompression in jaundiced patients with portal thrombosis secondary to chronic pancreatitis. Biliary endoscopic sterling provides

acceptable short and medium term resolution of common bile duct obstruction, but it can not be recommended as definitive therapy due to less acceptable long term results that are inferior to surgical treatment [71]. In one study, the presence of pancreatic head calcifications proved to be the main deterrent to success of endoscopic stenting [72]. Endoscopic stent, however, may be indicated or at least tried first in the presence of jaundice associated with cavernomatous transformation of the portal vein.

Internal pancreatic fistulas characterized by pancreatic ascites and\or pleural effusion secondary to leaking pseudocysts or disruption of the main pancreatic duct not properly walled off by inflammatory reaction, are well recognized complication of chronic pancreatitis 73,74,75. The diagnosis of internal pancreatic fistulas needs a high index of suspicion. The amylase levels will always be markedly elevated (>1000 Somogyi units/ 100 ml) and in the absence of hypoalbuminemia the albumin level will be > 3 g/ 100 ml. The serum amylase level of patients with internal pancreatic fistulas is not always raised. Endoscopic retrograde cholangiopancreatography has been considered to be the modality of choice in depicting the anatomy of the pancreatic ductal system and the internal pancreatic fistula site, and in the possible demonstration of fistulas tract and provides the road map to pancreatic duct, which is vital for the surgeon in deciding the appropriate surgical procedure. Therapeutic success of treatment of internal pancreatic fistulas may be achieved in 50-80% of patients with mild or moderate chronic pancreatitis after initial nonoperative management, but medical management should not exceed beyond 2-3 weeks 74. Patients with advanced disease have a much lower success rate of medical treatment and should be considered for early surgical intervention 73, 74.The surgical treatment of internal pancreatic fistulae will depend on the nature of the lesion demonstrated by direct ductal imaging by ERCP or by MRCP. Patients with a leaking pseudocysts or a disrupted dilated pancreatic duct will be best treated, respectively, by a cyst or a pancreatic duct internal drainage procedure, whereas distal pancreatectomy should be indicated for these patients with a narrow disrupted duct. External pancreatic drainage, avoiding loss of exocrine pancreatic function, may be a good alternative to resection in selected patients, but may be complicated by a persistent pancreaticocutaneous fistula in patients with proximal pancreatic duct obstruction 73 . More recently transpapillary pancreatic ductal stents have been used in patients who failed to respond to conservative measures 75. Desirable

prospective, randomized studies, comparing endoscopic and surgical management of internal pancreatic fistulae will certainly be difficult to conduct considering the diversity of ductal lesions present in this situation that makes patients difficult to compare.

The relevance of segmental portal hypertension for a patient with chronic pancreatitis is still poorly understood 76. Only transaction of the pancreas as in duodenum-preserving pancreatic head resection or proximal pancreatectomy will truly free the portal vein and superior mesenteric vein immediately. The removal of the majority of the inflammatory tissue around these vessels and a postulated regression of inflammation might also improve blood flow. However, complications from segmental portal hypertension are extremely rare. Its presence should therefore not influence the choice of an operation. In contrast complete portal vein obstruction with cavernomatous transformation was considered as contraindication for any effective surgical procedure for chronic pancreatitis. The risk of hepatic ischemia was considered too high to perform a partial pancreaticoduodenectomy. Extended drainage and limited resection procedures, in contrast, were shown to deliver control of symptoms and be safe at the same time 77.

RISK OF CONCOMITANT PANCREATIC CANCER:

The association of concomitant pancreatic cancer of the time of surgery of chronic pancreatitis is well known. The surgeon should bear this in mind both during initial evaluation of the patient with chronic pancreatitis as well as intraoperatively and subsequent evaluation for recurrence of pain, jaundice or weight loss should also bring suspicion of malignancy. Patients with chronic pancreatitis have an increased risk of developing pancreatic adenocarcinoma in relation to the general population. Two cohort studies demonstrate a strong correlation between chronic pancreatitis and an eventual diagnosis of pancreatic cancer, the cumulative risk of pancreatic cancer varying from 2.3 to 26.7% 78,79. In a multicenter historical cohort study of 2015 subjects with chronic pancreatitis, who were recruited from clinical centres in 6 countries, a total of 56 pancreatic cancers were identified during a mean follow-up time of 7.4 ± 6.2 years. The expected number of cancer cases calculated from country specific incidence data and adjusted for age and sex was 2.1, yielding a standardized incidence ratio of 26.3 (95% confidence interval, 19.9-34.2). For patients with a minimum of 2 or 5 years of follow-up, the respective standardized incidence ratios were 16.5 and 14.4. The cumulative risk of pancreatic cancer in subjects who were followed-up for atleast 2 years increased steadily, and 10 and 20 years after the diagnosis of pancreatitis, it was 1.8

and 4.0, respectively. Lowenfels et al concluded that the risk of pancreatic cancer is significantly higher in subjects with chronic pancreatitis and appears to be independent of sex, country and type of pancreatitis 78.

In patients with hereditary pancreatitis the estimated cumulative risk of pancreatic cancer approaches 40% and reaches almost 75% for patients with a paternal inheritance pattern 80.

Patients with tropical pancreatitis who develop pancreatic cancer differ from those in whom cancer occurs de novo in the presence of extensive calculi, higher frequency of diabetes mellitus, and presentation with abdominal pain rather than jaundice. Unlike de novo cancer, which has a predilection for the pancreatic head, Tropical pancreatitis cancer occur more frequently in the body and tail. About 60% of tumours appear to arise from an area in the body of the pancreas close to the neck. Once carcinoma supervenes, survival is short (median 11 months) despite resection of the tumour and chemotherapy 81.

Preoperative identification of cancer may be difficult in many cases. The occurrence of weight loss, appearance of constant upper abdominal pain, and deterioration in diabetic control should raise a suspicion of malignancy. Intraoperatively, the differential diagnosis may represent a formidable task considering the hardness of the enlarged pancreas present in both situations, which makes distinction by surgical palpation ineffective. Even in experienced centres approximately 10% of patients harbor a pancreatic carcinoma that is only diagnosed by histologic proof at the time of resection or after failure of drainage 29,34. In some reports, fine needle aspiration cytology proved to be of value for the diagnosis of pancreatic cancer 82, however only pancreatic resection or limited resection combined with external drainage can offer sufficient tissue material to accurately exclude malignancy 29,30,31. Needle and cytological biopsy specimens give false negative results in 20-30% and so are unreliable if they fail to show malignancy. In a report from Manheim surgical clinic, suspicion of cancer was the indication in 16.5% of patients operated upon for chronic pancreatitis 83. The increased incidence of pancreatic cancer in patients with chronic pancreatitis 78 further emphasizes this dilemma and accounts for some of the increases in proximal pancreatectomy for chronic pancreatitis.

Therefore, when there is strong suspicion of an underlying malignancy, pancreaticoduodenectomy should be considered in surgically fit patients. The risk of tumour spillage that may occur during the resective stage of the Frey or Beger procedures represents a drawback of these techniques and outweighs the potential benefits of a duodenum preserving resection for these patients.

Patients with Intraductal papillary mucinous tumours of the pancreas (IPMT) may present with symptoms similar to these of chronic pancreatitis. It is thus important to rule out the presence of an IPMT in patients referred for surgical treatment of chronic pancreatitis, particularly in lesions located in head of the pancreas. Endoscopic ultrasonography may be useful tool in these circumstances.

ENDOSCOPIC STENTING:

First it is important to realize that the natural history of pain confounds response to therapy. In Ammann's study 84, ~ 50% had intermittent pain and had a favorable course without surgery. If these patients had been submitted to a specific treatment (endoscopic, surgical, pancreatic enzymes, etc.), the favourable response would have been attributed to therapy. For example, there are two major endoscopic approaches: clearing stones from the pancreatic duct and/or stenting the pancreatic duct. Interestingly, by using the former approach, Cremer's group 85 reported that the best results (longest pain-free intervals) occurred in the patients who had no pain or few attacks of pain before endoscopic therapy. By performing a multivariate analysis of their data, they found that the best predictor of a long pain-free interval was infrequent attacks of pain before therapy. This analysis led them to state that "the best results are obtained if endoscopic therapy is performed early:, it is "often effective for years", and "propose endoscopic management early in the course of calcifying pancreatitis". Instead, an equally plausible explanation of their results is that the patients who "responded" to therapy would not have had attacks of pain regardless of treatment.

Similarly, these investigators interpreted results of their previous uncontrolled studies 86 that clearing of stones from the pancreatic produced pain relief in ~ 70% of patients. It is difficult to judge the effect of treatment in these studies because up to 71% of patients had relapsing pain and intervals between pain episodes varied from months to years. In addition, the frequency of painful attacks before treatment is not stated, the mean follow-up after treatment was short, and some patients had no or few attacks of pain. In another retrospective study, Smits et al, found that "endoscopic treatment of pancreatic stones is a valid approach in patients with pancreatic lithiasis" 87. However, they did not clear stones in 11 of the 53 patients and 3 of these 11 patients (27%) had complete

symptom relief; only 4 of 11 (36%) needed surgery; and 23% had treatment related complications (bleeding, exacerbation of pancreatitis, peroration, pancreatic abscess, stent clogging). It is unlikely that stenting of the pancreatic duct would have resulted in a similar improvement as compared to surgery because pain reduction after stenting only occurs in 54% of patients and may only be transient there is little change of pancreatic blood flow and the procedure results in parenchymal and permanent ductal changes in a number of patients. In a long-term prospective study, Adamek et al has shown that endoscopic pancreatic drainage and ESWL had no significant effect on pain and glandular insufficiency in patients with alcoholic chronic pancreatitis 88.

It is important to recognize that unproven treatments may be associated with significant complications and are expensive. For example, stenting the pancreatic duct, another proposed endoscopic treatment for pain, has an overall success of 85%, but pain reduction occurs in only 54% of the patients 89 and leads to parenchymal changes in 32% and no change in 5% of the patients after a mean of 192 days and concluded that stenting of the pancreatic duct was an experimental procedure. Of note, Cremer 85 hospitalizes patients 14 ± 14 days after the initial treatment, a very large expense.

A recent prospective, randomized trial comparing resection and drainage procedures with endoscopic stenting in a large number of patients with painful obstructive chronic pancreatitis demonstrated surgery to be more effective than endotherapy in long-term pain control 90.

NEURAL BLOCK:

Chemical or surgical neurotomies of pancreatic sympathetic pain afferents have been suggested as an alternative to operations on the pancreatic parenchyma in the management of patients with intractable pain from ‘small-duct’ chronic pancreatitis. The long-term results of percutaneous or endoscopic celiac plexus blockade are far from encouraging, since no more than 50% of the patients even develop a significant short-lived pain relief 91. A prospective, randomized study comparing the efficacy of EUS-guided versus CT-guided celiac plexus block failed to show any advantage of either approach 92. Bilateral thoracoscopic splanchnicectomy significantly decreased intensity of pain and improved quality of life 93. However, long-term pain relief was not often achieved and

pain recurred in approximately 50% of the patients. Favorable results of splanchnicectomy appear to be related to the response to differential epidural analgesia (DEA) as only patients with DEA-predicted sympathetic pain benefit from splanchnic neural ablation 94. The overall efficacy of this procedure also appears to depend on the patients to whom it is applied. In one study that included only patients with nonalcoholic pancreatitis, those who have had prior surgical or endoscopic interventions had a poorer response in pain relief compared to patients without prior intervention 95. On the contrary, two other studies involving mostly patients with alcoholic pancreatitis, found no correlation between the success rate of splanchnicectomy and the incidence of prior interventions directed at pain control 96, 97. Although bilateral thoracoscopic splanchnicectomy is safe, involves a minimal access approach, and may provide improved quality of life in patients with small-duct pancreatitis, further studies are needed to identify clearly the appropriate candidates for this procedure 95.

QUALITY OF LIFE AFTER SURGERY FOR CHRONIC PANCREATITIS:

The EORTC-QLQ has now been applied in two prospective randomized trials comparing surgical techniques in patients with chronic pancreatitis, which stress the draining or the resectional aspects of treatment to varying degrees 29,32. Statistically significant changes in functional and symptom levels were observed. In both trials, the patients' overall quality of life improved considerably. Relief of symptoms, especially of pain, fatigue, and loss of body weight, accounted for improvement of physical status, working ability and emotional and social functioning 29,31,32. These changes correlated well with the results of the separately assessed pain score and increase of body weight. The visual analog scale, as one feature of the pain score, has been shown to be a highly valid and reliable tool for measuring pain intensity (Ref.23,77 of SCNA). The additional information on frequency of pain attacks, analgesic regimen, and sick leave backs up the results of the visual analog scale. In comparison with McGill's pain questionnaire, the pain score had previously been tested in 19 patients suffering from pancreatic carcinoma, proving its validity and reliability.

AIM OF THE STUDY

The aim of the study was to analyse the long-term results of surgery for the patients who underwent surgery for chronic calcifying pancreatitis in a single institution during the 15 yrs period (January 1990 - December 2004) and followed up during two-year period from July 2003 to August 2005.

This is largely a prospective study that analyses the medium and long term outcome of surgery for chronic pancreatitis with particular emphasis on the quality of life for patients based on the mortality and morbidity rates associated with surgery, relief of symptoms, analgesic use, employment and long-term sequelae like exocrine and endocrine insufficiency of pancreatic surgery.

PATIENTS AND METHODS

Between 1990 and 2004, 151 patients were operated for chronic calcific pancreatitis at our department of Surgical gastroenterology, Government Stanley Hospital, Chennai. All these patients underwent pancreatic drainage or some form of

resectional procedures. Patients who have completed at least 6 months follow up included in the study. Patients who underwent pseudocysts drainage alone or bypass operations performed for bile or gastric drainage alone were excluded from this study. Sufficient information was available in 100 patients. The mean age at surgery was 35.6 ± 12.8 (Range 12 to 65 yrs). There were 70 Men 30 women. The diagnostic workup includes X- ray Abdomen erect, USG abdomen, ERCP and CT Abdomen. MRCP was done in selected cases. All of the cases of chronic pancreatitis were confirmed by histological examination.

Follow-up data were collected by periodic visits, telephonic interviews and information from family doctors. The records of all patients underwent drainage or resections for chronic pancreatitis at our department were reviewed. Preoperative date were reviewed included cause of chronic pancreatitis, clinical variables (symptoms, diabetes, and exocrine insufficiency), and morphological variables. Patients' characteristics of both alcoholic and tropical chronic pancreatitis were analyzed. Alcoholic chronic pancreatitis was defined as chronic pancreatitis associated with the consumption of greater than 50 units of alcohol per week for at least 5 years with no other etiological factor.

Preoperative variables examined were indication for surgery, type of pancreatic drainage or resection, morbidity and the mortality. The principal indication for operation was chronic pain, intractable to medical management associated with dilatation of the main pancreatic duct. Patient outcome was assessed by review of hospital inpatient and out patient records and telephone interview. Patients were assessed in the out patient clinic at 1 month, 3 months, 6 months and 1 year and annually thereafter. Data were recorded in a preprinted standard sheet with particular reference to operative morbidity and 30 day mortality rates; degree of pain relief, frequency and type of analgesia used, exocrine and endocrine insufficiency, weight gain, employment status, use of pancreatic enzyme supplements, alcohol consumption, hospital readmission, need for late reinterventions, co-existent medical disease, late mortality & causes of late death and quality of life assessment. The mean follow-up period was 43.5 months. The median follow-up period was 30 months.

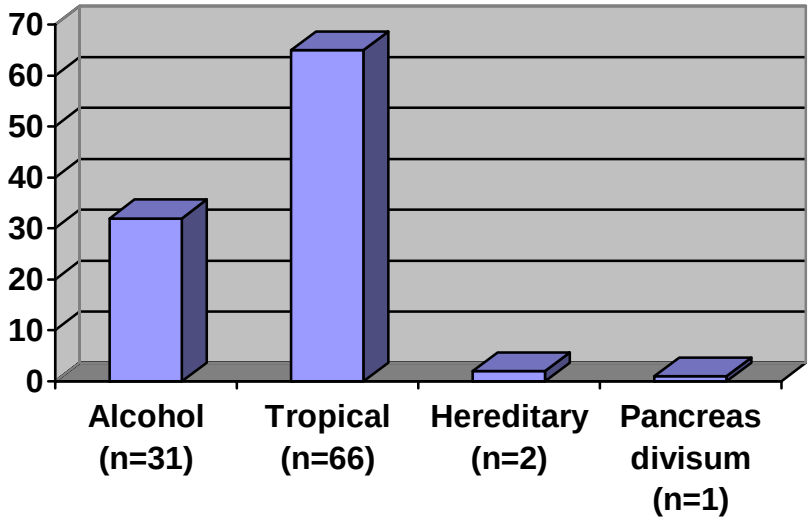
Complete pain control was defined as " no need of analgesic drugs" Partial pain control was defined as "need for minor analgesics" and no pain control was defined as "need of narcotics or hospital admissions". Diabetes mellitus was defined using World Health Organization criteria. Exocrine insufficiency was assessed clinically by the presence of gross steatorrhea at times

supplemented by pancreatic function tests. Categorical data were analyzed using Pearson's Chi – Squared test or Yates corrected Chi – Squared test or Fischer's exact test as appropriate and Odds ratio were given with 95% confidence interval. Continuous data were analyzed using student t – test.

RESULTS

Complete follow up data were available in 100 patients. 31 patients had alcohol pancreatitis, 66 patients had tropical pancreatitis, 2 patients had hereditary pancreatitis and one patient had pancreas divisum.

Bar diagram showing etiological classification:

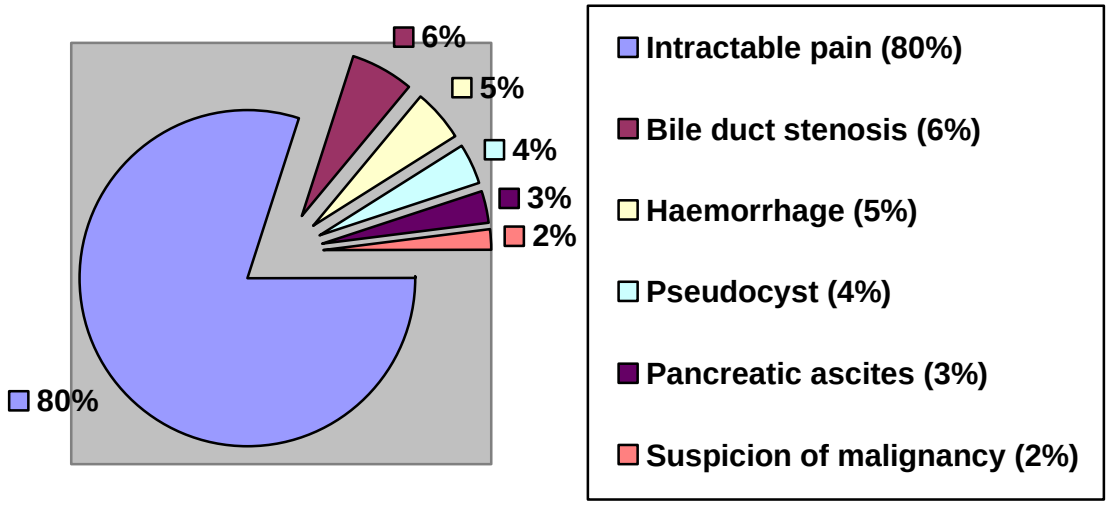


The median duration of symptoms before surgery was 34.66 months (range 1 month to 360 months).

In 80 patients (80 %) the principal indication for surgery was chronic intractable pain. Other indications were: bile duct stenosis in 6 patients (6%), haemorrhage in 5 patients (5%), pseudocyst in 4 patients (4%), suspicion of malignancy in two patients (2%) and pancreatic ascites in 3 patients (3%).

Among the cases listed above, gastrointestinal haemorrhage was caused by bleeding into a pseudocyst in 3 patients and one patient presented with haemoptysis and haematochezia. One patient presented with ruptured pseudocyst with gastrointestinal and intraperitoneal bleed.

Pie diagram showing indications for surgery:



Common associated symptoms included jaundice in 6 patients (6%) and gross exocrine insufficiency 17 patients (17%). 54 patients (54%) had endocrine insufficiency before surgery including 34 patients (34%) who were dependent on Insulin. 16 patients (16%) were taking regular daily oral analgesics, 84 patients (84%) were dependent on injectable analgesics for pain relief. 93 patients (93%) of working age were unemployed at the time of surgery.

On morphological analysis, 94 patients (94%) had main pancreatic dilatations, 98 patients (98%) had main pancreatic ductal calculi, 51 patients (51%) had parenchymal calcifications, 32 patients (32%) had parenchymal atrophy, 24 patients (24%) had inflammatory head mass and 10 patients (10%) had pseudocysts. Pseudocysts were single in all 10 patients. The site was at the pancreatic head region in 7 patients, body in 2 patients and tail in one patient. Pancreatic ductal strictures were seen in 9 patients and were at multiple sites in 5 patients, body in 2 patients, body and tail in one patient and one patient had stricture at head region.

Purulent fluid in pancreatic duct was present in six patients. Four of them were diabetics. MPD disruption was present in 3% of patients. One patient had disruption in the tail region. Two patients had disruptions in body of pancreas. Bile duct stenosis was present in 6 patients. All of them had smooth tapering terminal bile duct strictures.

Pancreatic abscess were seen four patients. Two patients had abscess in tail and body region. One patient had abscess in the body region and one had abscess in the tail region. One patient had mass in the neck region. One patient had associated necrosis of peripancreatic region around head. One patient had pancreas divisum, which was responsible for recurrent pancreatitis.

.Morphological findings are summarized in Table No. 1.

Table.No.1 The morphological characteristics in 100 patients:

Findings	Percentage
MPD dilatations	94%
MPD ductal calculi	98%
Parenchymal calcifications	51%
Parenchymal Atrophy	32%
Inflammatory Head Mass	24%
Pseudocysts	10%
Ductal strictures	9%
Multiple (n=5)	
Body (n=2)	
Head (n=1)	
Body & Tail (n=1)	
Bile duct stenosis	6%
Purulent fluid in MPD	6%
MPD disruptions	3%
Pancreas abscess	4%
Tail & Body	2%
Body	1%
Tail	1%
Mass Tail & Body	2%
Mass Neck	1%
Necrosis	1%
Pancreas divisum	1%

Apart from diabetes, significant co-existent medical diseases were seen in two patients including hypertension in one patient and another patient had pulmonary tuberculosis. 5 patients underwent endoscopic interventions. One patient had pancreatic sphincterotomy, one patient had extracorporeal shock wave lithotripsy (ESWL) with stent, one patient had CBD stenting and two other patients underwent pancreatic stenting. All these patients had poor control of pain following stenting. The patient who underwent ESWL with stent had developed pseudoaneurysm of splenic artery and subsequently had massive haemoptysis and haematochezia.

Among the surgical intervention, 91 patients (91%) underwent drainage procedures and 9 patients (9%) underwent some resectional procedures. Longitudinal pancreaticojejunostomy was performed in the majority of patients (n= 70). Pancreatogastrostomy was done in 4 patients. 13 patients underwent Local resection of head and longitudinal pancreaticojejunostomy (LR-LPJ / Frey's procedure). Puestow's procedure was done in two patients. One patient had excision of pseudo aneurysm, drainage of the pseudocyst and Roux-n-y pancreaticojejunostomy for gastro duodenal artery pseudo aneurysm bleeding into pseudocyst in head region. One patient had transcystic ligation of splenic artery pseudo aneurysm and cystogastrostomy for splenic artery pseudo aneurysm bleeding into pseudocyst in body region.

Among the resection group, 6 patients underwent distal pancreatectomy, and two patients had Whipple's resection for suspicion of head malignancy, which subsequently turned out to be chronic pancreatitis. Distal pancreatectomy, excision of pseudoaneurysm, closure of diaphragmatic and colonic rent and colostomy done for a case of splenic artery pseudoaneurysm eroding into diaphragm, bronchus and colon presenting with massive haemoptysis and haematochezia. One patient had excision of pseudoaneurysm and central pancreatectomy for splenic artery pseudoaneurysmal bleed. Table.2 summarizes the surgical interventions performed for 100 patients.

Table. 2. Surgical interventions adopted in 100 patients.

Interventions	Percent (%)
Drainage	**91**
Longitudinal Pancreaticojejunostomy	**71**

LPJ alone	57
LPJ + Choledochoduodenostomy	6
LPJ+ Cholecystectomy	3
LPJ + Pseudocyst / Ext. drainage	3
LPJ + enteroenterostomy	1
Transcystic excision of pseudoaneurysm + Cystogastrostomy + LPJ	1
LR-LPJ / Frey's procedure	**13**
Pancreatogastrostomy	**4**
PG alone	3
PG + Cholecystectomy	1
Puestow's procedure	**2**
Excision of pseudoaneurysm + Ext.drainage of Pseudocyst + Roux-n-y PJ	**1**
Resection	**9**
Distal pancreatectomy	**6**
DP alone	1
DP + LPJ	1
DP + Roux-n-Y PJ	1
DP + Excision of Pseudoaneurysm	2
Whipple's resection	**2**
Excision of pseudoaneurysm + Central pancreatectomy	1

ASSOCIATED COMPLICATIONS:

In 34 patients (34%), associated complications were identified before or during operation. 10 patients had associated Pseudocysts, 8 patients were treated with LPJ alone. cystogastrostomy was done in 1 patients and cystojejunostomy was done in one patient. Six patients who had terminal bile duct strictures were treated by biliary diversion i.e. choledochoduodenostomy along with

longitudinal pancreaticojejunostomy. 3 patients were presented with pancreatic ascites. Two patients were treated by Pancreaticogastrostomy alone. One patient had rupture of pancreatic duct in the tail region with pancreatic duct stricture in the head region. Distal pancreatectomy and Roux-n-y pancreaticojejunostomy was done for this patient. Four patients had associated pancreatic abscess. Three patients were treated with distal pancreatectomy and Roux-en-y pancreaticojejunostomy and one patient was treated with external drainage and LPJ. Five patients had associated gall stone disease and were treated with cholecystectomy. One patient had tuberculous ileocaecal mass and was treated with enteroenterostomy. Associated complications are shown in Table. 3.

Table.3 Management of complications associated with chronic pancreatitis

Complications	Pts	Management
Pseudocyst	10	8 LPJ alone
		1 LPJ + Cystogastrostomy
		1 LPJ + Cystojejunostomy
Bile duct stenosis	6	6 Choledochoduodenostomy
Haemorrhage	5	1 Excision of pseudoaneurysm + Ext.drainage off Pseudocyst + Roux-n-y PJ
		1 Transcystic excision of pseudoaneurysm + Cystogastrostomy
		1 Excision of pseudoaneurysm + Central pancreatectomy
		2 DP + Excision of Pseudoaneurysm
Pancreatic Ascites	3	2 Pancreaticogastrostomy
		1 DP with Roux-n-y PJ
Pancreatic abscess	4	3 DP + Roux-n-Y PJ
		1 LPJ + External drainage
Gall stone	5	4 LPJ + Cholecystectomy
		1 PG + Cholecystectomy
Ileocaecal mass	1	1 Enteroenterostomy

Patients' characteristics of both alcoholic chronic pancreatitis (ACP) and tropical chronic pancreatitis (TCP) were analyzed. Among the tropical chronic pancreatitis group, mean age was 31.6 years. Males: Females ratio was 1.76:1. Pain was present

in 97% of patients at the time of presentation. Jaundice due to biliary stricture was present in two patients; pseudocyst and pseudo aneurysm was present in four and two patients respectively. Mean pancreatic duct diameter was 9.1 mm; Ductal stones were present in 93.8% of patients. 13.6% of patients were presented with inflammatory head mass. Among the surgical intervention in TCP group 71.2% of patients underwent longitudinal pancreaticojejunostomy, 13.6% of patients underwent local head resection and longitudinal pancreaticojejunostomy (LR-LPJ or Frey's procedure), 3 patients had pancreaticogastrostomy, 2 patients had Puestow's procedure, 4 patients had distal pancreatectomy and one patient underwent central pancreatectomy.

Among the Alcoholic chronic pancreatitis group, mean age was 42.9 years which was significantly higher compared to TCP group (31.9). All were males. 93.5% of patients were presented with pain. 6.5% of patients had jaundice. Pseudocysts and pseudoaneurysm were present in six and two patients respectively. Suspicious of malignancy was present in two cases. Mean pancreatic duct diameter was 9.3 mm which is similar to TCP group. Inflammatory head mass was present in 22.6% of patients. Ductal stones were present in 80.6% of patients. 67.7% of patients underwent longitudinal pancreaticojejunostomy. 13% of patients had LR-LPJ. One patient had pancreaticogastrostomy. One patient had excision of pseudoaneurysm and Roux-n-y pancreaticojejunostomy. Two patients had distal pancreatectomy. Two patients were subjected to Whipple's resection based on the suspicious of malignancy. In a comparative analysis, patients in tropical chronic pancreatitis were

significantly younger than in alcoholic chronic pancreatitis. Pseudocysts were significantly more common in the alcoholic chronic pancreatitis. Suspicion of malignancy is significantly more common as an indication for surgery in the alcoholic pancreatitis. The comparative analysis is shown in Table.4.

Table 4. The comparative analysis of TCP and ACP

Parameters	TCP	ACP	P Value

No. of patients	66	31	-
Age (mean ± s.e.m)	31.9 ± 12.9	42.9 ± 8.1	**0.001**
Sex (Male: Female)	37:29	All Males	-
Pain	64 (97%)	29 (93.7%)	0.4
Complications			
Biliary stricture	2 (3%)	3 (9.7%)	0.4
Pseudocyst	4 (6%)	6 (19.3%)	**0.04**
Pseudoaneurysm	3 (4.5%)	2 (6.5%)	0.5
Suspicious of malignancy	0	2 (6.5%)	**0.04**
PD diameter (mm) (mean)	9.1	9.3	-
Ductal stones	61 (93.8%)	25 (80.6%)	0.09
Inflammatory head mass	9 (13.6)	7 (22.6%)	0.27
Surgical intervention			
Drainage procedures			
LPJ	47 (71.2%)	21 (67.7%)	0.73
LR-LPJ	9 (13.6%)	4 (13%)	0.6
PG	3 (4.5%)	1	0.6
Others	0	1	-
Resections			
Distal pancreatectomy	4 (6%)	2 (6.5%)	0.4
Whipple's	0	2	**0.04**
Central pancreatectomy	1	0	-

EARLY OPERATIVE MORBIDITY AND MORTALITY:

There was no difference between hospital mortality (1.4% Vs 0%) between drainage and resection groups. Significant morbidity after pancreatico -jejunostomy included pancreatic fistula (2%), delayed gastric emptying (1%), and gastrointestinal hemorrhage due to pancreatico jejunostomy anastomotic bleeding (1%). The latter patient

required reoperation. Patient died on d 24. Among resectional procedures, one patient had pancreatic fistula following distal pancreatectomy and one patient had biliary peritonitis due to bilioenteric anastomotic leak following Whipple's resection. The latter required reoperation and fared well after second surgery. Two patients in the drainage group and one patient in resection group developed pulmonary complications - atelectasis. One patient had delayed gastric emptying after distal pancreatectomy, which resolved with conservative treatment. One patient developed subphrenic abscess following distal pancreatectomy, which was treated with ultrasound guided drainage and antibiotics. Six patients in the drainage group and four patients in resection group had minor wound infection and one patient had urinary tract infection following LR-LPJ.

There were a total of 24 complications in 18 patients (morbidity 18%) within the postoperative period. The presence of a pancreatic fistula was defined by the drainage output of greater than 50 ml abdominal fluid per day with an amylase content of greater than 3 times the serum value (> 300 units/ liter). Non operative management was used for pancreatic fistula and drain track infection. Two patients required reintervention. One patient required a relaparotomy for gastrointestinal bleed due to pancreaticojejunal anastomotic bleed and subsequently died.

Another patient required relaparotomy for bile peritonitis and subsequently recovered. 30-day In-Hospital mortality was 1%. Postoperative complications and their management are shown in Table 5.

Table.5. Postoperative Complications and management

Complications	Drainage	Resection	Management
Pulmonary			
Atelectasis	2	1	Antibiotics
Gastrointestinal			
Pancreatic fistula	2	1	Conservative
GI Hemorrhage	1	-	Reoperation
Delayed gastric emptying	1	1	Conservative
Subphrenic/ Pelvic abscess	-	1	Antibiotics And USG guided Drainage
Bile leak	-	1	Reoperation
Others			
Septicemia	-	2	Antibiotics
Wound infection	6	4	Conservative
UTI	1	-	Antibiotics

In a comparative analysis between drainage and resection group, hospital morbidity was significantly higher in resection group (p= 0..01, OR = 7.5, 95% CI = 1.5-39). No significant difference was found in hospital mortality and reoperation. The operative procedures performed and hospital course after each type of surgery are summarized in the Table 6.

Table 6. Operative Procedures Performed and Hospital Course

Procedures	No. of Patients	Hospital Mortality	Hospital morbidity	Mean Hospital stay	Reoperation (no.)	Mean follow -up (months)
Drainage						
LPJ	71	1(1.4%)	7(9.8%)	11.3	2	47
Frey's	13	-	5(38.5%)	13.5	-	17.1
PG	4	-	-	10.8	-	31
Puestow's	2	-	-	16.0	-	144
Excision of PA+ Roux-n-y PJ	1	-	1(1/1)	43.0	-	18
Resection						
DP	6	-	3(50%)	11.8	-	40.5
Whipple	2	-	2(2/2)	16.0	1	54.0
Central pancreatectomy	1	-	-	10.0	-	24.0

RELIEF OF PAIN:

Long-term pain relief was good in 87 patients (87 %), fair in 11patients (11 %) and poor in 2 patients (2 %). The results of the different types of operation on long-term pain relief are presented in Table 7. The highest rate of pain relief was achieved after LR-LPJ or Frey's procedure (92.3%), followed by distal pancreatectomy (85.7%) and Longitudinal pancreatico jejunostomy (83.1%). However, when compared, differences between the various surgical procedures concerning pain relief were not significant.

Of 81 patients who had been operated for intractable pain, on follow-up discounting early and late deaths, 65 (84%) of the 77 surviving patients had complete relief of pain and 8 (10%) patients had better control of pain and able to attend to their normal preillness daily routine. 4 (5%) patients had persistent epigastric pain requiring analgesics and not satisfied with the results of surgery. There was no significant difference in the pain relief between drainage and resection group (p= 0.73). Table.7 summarizes the degree of pain relief after various procedures for chronic pancreatitis.

Table 7. Pain relief in patients undergoing surgery for chronic pancreatitis

Operative Procedures	Pain Relief		Mean follow –up (months)
	No. of Patients	Percentage (%)	
Drainage			
LPJ	59/71	83.1	47
Frey's	12/13	92.3	17.1
PG	4/4	3/4	31
Puestow's	1/2	1/2	144
Excision of PA+ Roux-n-y PJ	1/1	1/1	18
Resection			
DP	5/6	83.3	40.5
Whipple	2/2	1/2	54.0
Central pancreatectomy	1/1	1/1	24.0

PANCREATIC INSUFFICIENCY:

The overall insulin requirement was decreased significantly after operation both at 3 months and at 3.6 years.. Two of thirty-three patients who had diabetes were able to stop insulin therapy and control their diabetic status with diet alone and four of patients had obtained euglycemic status and diabetic free status. 18 of 34 patients who were dependent on insulin could be able to stop insulin therapy and control their diabetic status with oral hypoglycemic agent therapy (OHA) alone. There was significant reduction in the requirement of insulin dose after surgery (18.6 Vs 30.8, $p < 0.001$). There was a significant decrease in fasting and postprandial blood glucose levels in diabetic patients after surgery, 147.6 Vs 204.8 and 232.4 Vs 325.3 respectively. Table 8 summarizes the dose of insulin required to control the diabetes and the blood glucose levels on follow-up.

Table.8. The insulin requirement and blood glucose levels in 54 diabetic patients subjected for surgery at mean 3.6 years follow-up

Parameters	Before surgery	After surgery	P value
Units/day per patient, mean(± SE)	30.8 (3.8)	18.5 (3.4)	0.001
Fasting (mg/dl)	204.8 (11.5)	147.6 (8.1)	0.001
Postprandial (mg/dl)	325.3 (16.8)	232.4 (12.2)	0.001

Pancreatic insufficiency was slightly improved in patients subjected for longitudinal pancreatico jejunostomy group, while it was not changed in LR-LPJ group. Among the resection group, endocrine insufficiency worsened in patients who had distal pancreatectomy and unchanged in other operations. Steatorrhea improved in six of 17 patients after surgery and remained unchanged

in four. Four new cases of malabsorption were detected postoperatively in addition. Pancreatic insufficiency after surgery was summarized in Table 9. There was no difference in pancreatic insufficiency between resection and drainage group.

Table 9. Pancreatic Insufficiency after Surgery for Chronic Pancreatitis

PROCEDURES	PANCREATIC INSUFFICIENCY								MEAN FOLLOW-UP (months)
	Diabetes				Steatorrhea				
	Preop		Postop		Preop		Postop		
	No. of pts	(%)	No. of pts	(%)	No. of pts	(%)	No. of pts	(%)	
Drainage									
LPJ	41/71	57.7	33/71	46.5	9/71	12.7	5/71	7.0	47
Frey's	7/13	53.8	7/13	53.8	3/13	23.1	3/13	23.1	17.1
PG	1/4	25.0	1/4	25.0	2/4	-	0	-	31
Puestow's	2/2	2/2	1/2	1/2	0	-	0	-	144
Excision of PA+	1/1	1/1	0	0	0	-	0	-	18
Roux-n-y PJ									
Resection									
DP	1/6	16.7	1/6	16.7	1/6	16.7	0	0	40.5
Whipple	1/2	1/2	1/2	1/2	2/2	2/2	2/2	-	54.0
Central pancreatectomy	-	-	0	0	0	-	0	-	24.0

On follow-up, there was significant reduction in number of patients experiencing pain after surgery (96% Vs 21%, p= 0.001). Similarly, there were significant reduction in number of patients requiring injectable analgesics (87% Vs 7%, p= 0.001), number of patients requiring enzyme replacement (80% Vs 54%, p= 0.001), number of patients employed (9% Vs 82%, p= 0.001) and the number of patients dependent on insulin (34% Vs 13%, p= 0.001). But there was no significant change in diabetic status and the

number of patients having steatorrhoea. Table. 10 summarizes overall results on pain relief, endocrine and exocrine insufficiency and employment status.

Table. 10. Overall outcome of surgery for chronic calcific pancreatitis.

	Grand Total		P value [OR (95% CI)]
	Before Surgery (%)	After Surgery (%)	
Pain	96	21	0.001 [90 (28-329)]
Injectable Analgesics	84	7	0.001 [70 (25-201)]
Requirement for Enzyme Replacement	80	54	0.001[3(2-7)]
Diabetes	54	44	0.12
Steatorrhoea	17	10	0.15
Employed	9	82	0.001[47(18-120)]
Insulin Dependency	34	13	0.001[3(2-8)]

The Alcohol withdrawal rate was 90.3%. There was a significant reduction in alcohol consumption after surgery with 3 out of 31 (9.7%) of patients was continuing to drink alcohol beyond 1 year after surgery ($p < 0.0001$). There was significant difference in pain score between those who continued to drink and those who abstained. Similar to overall results, the subgroup analysis also showed significantly improved results in both alcoholic and tropical chronic pancreatitis group.

In Alcoholic chronic pancreatitis, there was improvement in number of patients experiencing pain (93.5% Vs 22.6%, p= 0.001), number of patients requiring injectable analgesics for pain relief (80.6% Vs 6.5%, p= 0.001), number of patients requiring pancreatic enzyme replacement (90.3% Vs 64.5%, p=0.01), number of patients having diabetes (64.5% Vs 51.6%, p= 0.01), number

of patients employed (12.9% Vs 77.4%, p= 0.001) and number of patients dependent on insulin (33.5% Vs 12.9%, p= 0.05). There was no significant change in number of patients having steatorrhoea (25.8% Vs 9.7%, p=0.78).

In tropical chronic pancreatitis, there was significant improvement in number of patients experiencing pain (97% Vs 19.7%, p= 0.001), number of patients requiring injectable analgesics for pain relief (86.4% Vs 6.1`%, p= 0.001), number of patients requiring pancreatic enzyme replacement (74.2% Vs 47%, p=0.01), number of patients employed (7.6% Vs 83.3%, p= 0.001) and number of patients dependent on insulin (31.8% Vs 12.1%, p= 0.05). There was no significant change in number of patients having diabetes and steatorrhoea. Outcome analysis, according to etiological classification was summarized in Table 11.

Table 11. Outcome of Surgery in chronic calcific pancreatitis according to etiological classification. Data were expressed as number of patients.

	Alcoholic pancreatitis (31)				Tropical Pancreatitis (66)			
	Before surgery (%)	After surgery (%)	P Value	OR (95 % CI)	Before Surgery (%)	After surgery (%)	P Value	OR (95 % CI)
Misused alcohol	31 (100)	3 (9.7)	0.001	-	-	-	-	-
Pain	29 (93.5)	7 (22.6)	0.001	49 (8 - 399)	64 (97)	13 (19.7)	0.001	130(26 – 887)
Injectable Analgesics	25 (80.6)	2 (6.5)	0.001	60 (10–500)	57 (86.4)	4 (6.1)	0.001	98 (26 -420)
Requirement for Enzyme Replacement	28 (90.3)	20 (64.5)	0.01	5 (1 – 27)	49 (74.2)	31 (47)	0.001	3 (2 - 7)
Diabetes	20 (64.5)	16 (51.6)	0.01	5 (1 – 27)	33 (50)	26 (39.4)	0.22	-
Steatorrhoea	8 (25.8)	3 (9.7)	0.78	-	9 (13.6)	7 (10.6)	0.59	-
Employed	4 (12.9)	24 (77.4)	0.001	23.1 (5 – 115)	5 (7.6)	55 (83.3)	0.001	61 (18 -223)
Insulin Dependency	11 (35.5)	4 (12.9)	0.05	3 (1 – 15)	21 (31.8)	8 (12.1)	0.01	3 (1- 9)

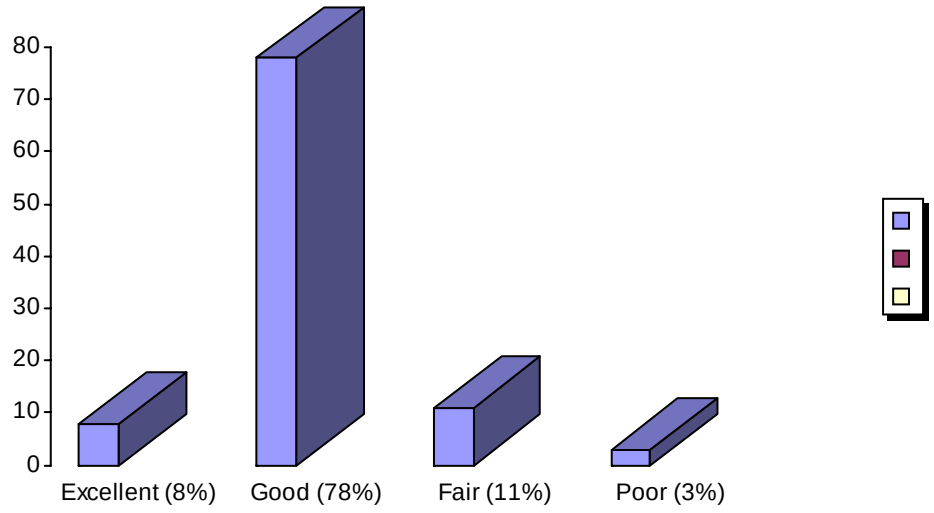
Patients were considered to be in good health had lower alcohol and regular analgesic requirements for insulin and pancreatic enzyme supplementation. Patients considered to poor health category had continued use of alcohol and analgesic use and high incidence of endocrine and exocrine insufficiency characterized this group. Over all health assessment was classified as

excellent in 8 patients (8%), good in 78 patients (78 %), fair in 11 (11%) and poor in 3 (3%). Overall health status is shown in Table.12.

Table.12. The Overall health status on follow-up in 100 patients

Follow-up (Months)	Functional Results			
	Excellent	Good	Fair	Poor
6-24	4	39	2	3
25-60	2	28	1	-
61-120	1	6	8	-
121-180	1	5	-	-
Total (%)	8(8%)	78(78%)	11(11%)	3(3%)

Diagram showing the health status:



QUALITY OF LIFE

Although no formal instrument measuring quality of life was used, quality of life after surgery was inferred from the results graded both by degree of pain relief and activity status. The median number of hospital admission fell after surgery for all procedures from 2 admissions per year to none by 12 months after surgery which was maintained during follow-up. There was a significant increase in those in regular employment after surgery. 82 of 86 (95.3%) patients rated as in good health were employed (full time), whereas only 6 of 11 (54.5%) in fair health and none in poor health were employed. The proportion of patients able to work a function normally after surgery increased markedly (9% vs. 82%, P<0.0001); 79% of patients unable to work or to function normally prior to surgery were able to do so after surgery.

WEIGHT MAINTENANCE OR GAIN

87 Patients gained weight during the follow-up. The mean weight gain was 3 Kg. Five patients maintained their weight. Eight patients had weight loss with the mean of 6.2 Kg. There was significantly weight gain in the postoperative period. (p= 0.001,OR= 77, 95% CI = 28-220).

RE - HOSPITALIZATIONS:

19 patients required one or more hospital admissions during follow-up. 9 patients admitted for pain abdomen related to acute exacerbation of chronic pancreatitis, which was treated conservatively. Two patients admitted with diabetes mellitus related problems. Three patients had oesophagitis/gastritis related problems. One patient had been admitted for repair of incisional hernia. Three patients had mortality owing to malignancy of head of pancreas. These patients had pain abdomen consequence of unsuspected malignancy intraoperatively. One of these patients had gastric outlet obstruction symptoms. All these patients had biopsy proven

malignancy and had disseminated malignancy at the time of diagnosis. Two patients died 11/2 years and one patient died at 1 year after surgery. One patient had ruptured splenic artery pseudo aneurysm, gastric perforation, splenic infarction, necrosis of pancreatic parenchyma and colonic perforation. This patient was operated after resuscitation. Distal pancreatectomy, Splenectomy, excision of pseudo aneurysm, necrosectomy, closure of gastric perforation and transverse colostomy was performed. The patient was on prolonged postoperative ventilator support and subsequently died. Reasons for re - hospitalization are listed in Table. 13.

Table. No.13. Re - hospitalizations details:

Reasons	No. of patients
Admission related to chronic pancreatitis	
Pain abdomen	9
Diabetes	2
Unsuspected / Missed Malignancy	3
Admission for other conditions	
Oesophagitis/Gastritis	3
GI Bleed	1
Incisional Hernia	1

DISCUSSION

According to the conventional wisdom, an operative procedure for chronic pancreatitis, as a palliative measure on an already functionally impaired gland, it should be as conservative as possible to limit the occurrence especially of endocrine failure.

Contrary to this, aggressive approach has been advocated by some series. Berney et al [3] reported pancreatic resection for severe chronic pancreatitis in 68 patients and shown that resection was safe and had long-term results of pain control.

Our series, based on the conventional concept, to preserve as much functioning parenchyma and islet cells as possible, only nine patients were subjected for resectional procedures. 8 of the 9 patients had pain relief; four of nine patients had endocrine insufficiency.

Longitudinal pancreatico jejunostomy (LPJ) is safe. We had one postoperative death with LPJ (mortality 1%). Many authors have reported low mortality rate (0 %) [10]. During follow up, 3 patients (3%) died at a mean of thirteen months after the operation, which is comparable with reported rate [11]. Pain relief was classified as good in 65 of 77 patients (84%) who underwent surgery for intractable pain.

Our study showed that operative intervention (both drainage and resection) can achieve pain relief and a better quality of life in selected patients. Frey's procedure appears to achieve highest pain relief (92.3%), Careful patient selection presumably based on structural changes within the pancreas and adjacent organs is the key factor in achieving good results. Although our group fully accepts the concept of 'burn out' of the disease leads to eventual spontaneous relief of the chronic pain. In our study, operative therapy not only led to long lasting pain relief in the majority of patients operated, but also resulted in a significant increase in the proportion of patients able to work or function normally in a society from 9% to 82%, according to a better quality of life.

In our study, among the alcoholic chronic pancreatitis, 4 of 31 patients (12.9%) continued to consume alcohol. Most of these patients fell into poor or fair health categories. Despite uncertainty as whether abstinence of alcohol truly results in diminished pain, there is little debate that abstinence should be recommended. Lankisch et al and Layer et al demonstrated that only 13% to 20% of deaths in patients with chronic pancreatitis are directly related to pancreatitis 6, 7. The majority of deaths in these patients occur as a result of diseases that are related to tobacco or alcohol.

Although higher proportion of patients with tropical chronic pancreatitis patients had pain relief following surgery, we did not find any correlation between the pain relief in alcoholic pancreatitis and those who were not alcoholic. This result is similar to that reported by Brinton et al 40 who reported results of lateral pancreaticojejunostomy in 39 patients. This is in contrast to report by Sato et al, who found that over mean period of 9.1 years only 50% of patients with alcoholic pancreatitis had a good result after surgery compared to 83% of those who had nonalcoholic pancreatitis 41. Another report by Sharma et al 42 found that 88% of patients of patients with nonalcoholic chronic pancreatitis had immediate and lasting pain relief.

Studies reporting the incidence of alcohol abuse and results of surgery after pancreaticojejunostomy for chronic pancreatitis are shown in Table. 14.

Table.14. Incidence of alcohol abuse and results of surgery after pancreaticojejunostomy for chronic pancreatitis.

Study	Year	No.	Alcohol abuse (%)	Operative mortality (no.) (%)	Late deaths (No.) (%)	Relief of pain		
						Poor	Fair	Good
Greenlee et al	1990	100	100	4 (4)	3 (3)	22	16	37
Hakaim et al	1994	50	60	0 (0)	5 (10)	2	24	60
Sharma et al	1998	58	0	4 (7)	4 (7)	8	12	79
Present study		100	31	1 (1)	3 (3)	5	10	84

In our study, the type of surgery or the etiology of chronic pancreatitis did not influence the long-term outcome of patient with chronic pancreatitis. This could be partly explained by the nature of the disease. The etiology and perception of pain in chronic

pancreatitis is definitely multi-factorial and it is influenced by a variety of factors including the social and cultural dynamics, co-morbid illnesses and continued use of alcohol.

Our group of patients who had poor relief of pain, continued to indulge in alcohol consumption and because alcohol withdrawal could not be obtained in those patients, we have not employed operative strategies for this set of patients.

When the health status of the patient was analyzed in greater details, health status was considered Excellent in 8 (8%), good in 78 (78%), fair in 11 (11%) and poor in 3(3%) of patients who were alive at review.

ENDOCRINE AND EXOCRINE INSUFFICIENCY:

The operation resulted in an improvement of patient's diabetic status, with about 24 of 33patients (72.7%) who were dependent on insulin before surgery, being able to stop taking insulin and euglycemic status was achieved with oral hypoglycemic drugs or by diet alone. Exocrine pancreatic dysfunction was improved in 50% of patients who had steatorrhea preoperatively.

Similar experiences have been reported from India in patients with tropical calcific pancreatitis. Sidhu et al have shown that there was significant improvement of endocrine insufficiency and slight improvement of exocrine insufficiency in patients with tropical chronic pancreatitis undergoing modified Puestow procedure in a long-term follow-up study 17. Similar experience from Western studies by Nealon et al also showed that occurrence of endocrine and exocrine insufficiency can be delayed by longitudinal pancreaticojejunostomy 16. In our study, there was no difference in pancreatic insufficiency between and resection group which is similar to a report from Mayo clinic study by Sakorafas et al 28.

ASSOCIATED COMPLICATIONS:

In 14 patients (14%), LPJ was combined with another procedure to manage a complication of chronic pancreatitis. 10 patients (10%) had associated pseudocysts. 8 patients with pseudocysts were managed with longitudinal pancreaticojejunostomy alone. This study further supports the concept of Nealon et al that most pseudocysts located in the head and body of the gland can be incorporated into the LPJ 11.

6 Patients (6%) had terminal biliary stenosis managed by biliary enteric diversion: i.e. Choledocho duodenostomy. The incidence was similar to that reported in literature 68. 3 Patients had pancreatic ascites, which was initially managed conservatively. Two required pancreaticogastrostomy and another required distal pancreatectomy and splenectorny. It is not necessary to attempt excision of the fistulous track because it will close spontaneously after adequate decompression of the pancreatic duct. This experience emphasizes the common occurrence of associated complications that must be considered in the preoperative evaluation and when possible, corrected during the surgical procedure. We had three late deaths related to unsuspected pancreatic cancer. These patients presented with markedly unrelieved pain in the follow-up. This incidence is similar to that reported by Markowitz et al in which they have shown that unsuspected or missed malignancy of head of pancreas is an important cause of persistent pain after decompression procedure 34. This further emphasizes that one should have a high index of suspicion of pancreatic cancer in patients presenting with head mass.

Patients whom underwent endoscopic intervention had poor control of pain and this modality may be associated with major morbidity and life threatening complication. In our study, we had one major complication, splenic artery pseudoaneurysm with diaphragmatic and bronchial erosion and presenting with haemoptysis following ESWL and pancreatic stenting and other patients who underwent stenting also had poor short-term results.

QUALITY OF LIFE AFTER SURGERY FOR CHRONIC PANCREATITIS:

Although no formal instrument measuring quality of life was used, quality of life after surgery was inferred from the results graded both by degree of pain relief and activity status. There was significant decrease in number of hospital admissions after

surgery (2 versus none, $p < 0.05$), and significant increase in regular employment and able to work a function normally (9% versus 82%).

FUTURE DEVELOPMENTS:

Genetic abnormalities in chronic pancreatitis:

It is now well established that there are genetic abnormalities in chronic pancreatitis. The specific gene mutations associated with different forms of chronic pancreatitis, include:

Cationic trypsinogen (PRSS1) gene mutation in hereditary pancreatitis.

Serine protease inhibiter (Kazal type 1 (SPINK1)) gene mutation in idiopathic chronic pancreatitis. SPINK 1 gene mutation has also been reported in tropical and alcoholic chronic pancreatitis.] Cystic fibrosis transmembrane regulator gene mutation (CFTR) in chronic pancreatitis associated with cystic fibrosis.

Genetic studies in the diagnosis of chronic pancreatitis:

Conventionally, hereditary chronic pancreatitis is diagnosed only when one or more members of the family are affected by this criterion, a number of cases of hereditary chronic pancreatic can be missed because it may occur even when no other family has the disease. This group is thus labelled idiopathic chronic pancreatitis. However, PRSS 1 gene mutation has been detected in approximately 10% of patients with idiopathic chronic pancreatic without a family history of chronic pancreatitis. Since PRSS 1 gene mutation occurs in hereditary chronic pancreatitis, these patients with a negative family history but evidence of the gene mutation should be labelled hereditary chronic pancreatitis [98]. Another 10% of patients with idiopathic chronic pancreatitis have the R122 H trypsinogen gene mutation. These patients get the diseases much earlier (mean age 14 ± 3 years) compared with those without the R122 H trypsinogen gene mutation (mean age 38 ± 2 years).

Genes as potential markers and target in chronic pancreatitis:

In 2002, a near complete sequencing of the human genome99 was achieved. This allowed, with the help of novel technologies such as DNA array, the simultaneous analysis of a large number of genes and the identification of disease-specific genes. The first study using DNA array technology in CP was conducted by Fries et al.100. On screening 5600 human genes, 6 genes specific to CP were identified. These showed a significantly increased expression in CP compared to the normal pancreas as well as pancreatic cancer.100 These 6 genes included the cartilage oligomeric matrix protein (COMP), the cysteine-rich secretory protein-3 (CRISP3) and tryptase.10

Tryptase is a serine protease, stored in the mast cells, which is known to induce the synthesis of type 1 collagen by human fibroblasts and stimulate fibroblasts proliferation and chemotaxis. Mast cells are well-known effector cells of immediate-type allergic reaction. A cross-linking of receptor-bound IgE on the mast cells membrane by specific allergens leads to the release of preformed

mediator. Though these and other mechanisms, mast cells exert their role in allergic reactions as well as in acute and chronic inflammatory settings. In CP, there is a significant increase in the total number of mast cells compared to the normal pancreas101. Interestingly, there is also a correlation between the number of mast cells, and the extent of fibrosis and intensity of inflammations101. Furthermore, IgE-dependent mast cells activation is higher in CP than in normal pancreas; however, there is no difference in the number of mast cells or IgE- positive mast cells between CP of different aetiologies. In CP, mast cells are mostly located in the fibrous tissue and around the regenerating ducts, which are positive for the c-kit receptor----, the receptor for the mast cells growth factor SCF (stem cell factor). 101 It is thought that mast cells are a relevant component of the inflammatory infiltrate in CP, which might play a role in tissue destruction and remodeling in this disease.

The CRISP3 is a member of the cysteine-rich secretory protein family, detected in several human tissues, predominantly in the salivary gland, pancreas and prostate. In addition, CRISP3 is considered a defence-associated molecule in mammals. In CP, there is an increased expression of this molecule, which was mainly localized in the cytoplasm of dedifferentiating acinar cells and in tubular complexes100 Interestingly, CRISP3 expression was weak to absent in cancer cells, and normal acinar and ductal cells in pancreatic cancer as well in the normal pancreas. 100 The predominant localization of CRISP3 in Acinar cells dedifferentiating into tubular complexes as well as in these tubular structures itself suggests that this molecule has some role in the pathophysiology of CP.

The third gene which is selectively upregulated in COMP. COMP is a member of the thrombosponding family of extracellular matrix proteins. It was discovered initially in the cartilage of several human organs. In CP, expression of COMP was evident in the cytoplasm of the degenerating acinar cells, but not in the tubular complexes, or in normal acinar or ductal cells. 101 In additions, COMP is also present in the fibrotic tissue in CP. Preferential expression of COMP in the dedifferentiating acinar cells in CP suggests a potential role of this gene in causing degeneration and dedifferentiation of the Acinar cells.101 COMP might thus serve as a marker for tissue destruction and disease activity in CP.

Tropical Chronic Pancreatitis:

While the molecular basis of alcoholic and idiopathic CP has been extensively studied, it has not been compared with tropical CP.102

Among various causes, nerve alteration and neuroimmune interaction have been suggested to participate in causing pain in CP. The German group compared neural changes and the pattern of Perineural inflammatory cell infiltrates in three different aetiological forms of CP (alcoholic, idiopathic and tropical) and evaluated whether there were any differences between them. In all samples, the number of nerves, area of neural tissue, nerve size, and percentage of neural issue and perineural inflammatory cell infiltrates were analyzed histologically. In comparison with normal controls, all these parameters were significantly increased in all three forms of CP except the number of nerves in tropical CP. Despite this observation, the percentage of neural tissue was similar in all the three forms studied and this was primarily due to the fact that while the number of nerves was not increased in tropical CP, the size of the individual nerves was bigger in tropical CP than alcoholic and idiopathic CP. When the degree of perineural inflammation was evaluated, no difference was observed among the three groups. Thus, it was concluded that independent of the underlying aetiology, CP is associated with an increase in neural tissue and neural alterations occur in a similar fashion irrespective of the type of initiating event 103.

There is increasing evidence that immune mechanisms may be crucial in the development alcoholic CP. However, it is not known whether difference in the underlying aetiology influence the inflammatory reaction in patients with CP. In the second part of our study, the histological feature and the pattern of inflammatory cell infiltration were studied in three groups of CP. Haematoxylin and eosin-stained tissue sections were used for histological evaluation. For immunohistochmical characterization of the inflammatory reaction, 4 antibodies--- CD4, CD8, CD45 and CD68----were used. Quantitative evaluation of the various cell infiltrates was performed with computer -assisted image analysis. The inflammatory cell infiltration pattern was also evaluated. It was observed that the degree of endophlibitis and the overall density of plasma cells was greater in tropical CP than alcoholic CP. The grade of intralobular fibrosis was significantly higher in tropical CP compared with idiopathic CP. No significant quantitative difference in the specific cellular infiltrates (CD4, CD8, CD45, CD68) were observed in the three groups. It was thus concluded that different aetiological forms of CP result in similar histological feature and a comparable inflammatory cell reaction indicating that the disease,

independent of the underlying aetiology, reaches a common immunological stage beyond which it appears to progress as a single distinctive entity.104 Additional information is now available on tropical CP. Chandak et al. Have demonstrated the importance of mutations in the *SPINK1* gene in tropical CP.105 In India, mutation in the *SPINK1* rather than the *PRSS1* gene are significantly associated with tropical CP.105

Genetic Basis of Pain in Chronic Pancreatitis:

Increased intraductal pressure as a result of single or multiple stricture and/or calculi is believed to be the cause of pain in patient with dilated main pancreatic duct disease. The other causes of pain are duodenal and common bile obstruction due to ongoing pancreatic inflammation and scarring. However, at the molecular level , the most recent concept of the pathogenesis of pain in CP postulates alteration in the pancreatic nerves and neuroimmune interactions as crucial in the development of Pin syndrome.106 In addition to the nerve changes described above in tropical CP, inflammatory cells have also been implicated in neural changes in CP, since infiltration of the pancreatic nerves by immune cells and destruction of the perineurium have correlated with the intensity of pain in CP.107 The recruitment of inflammatory cells in to the inflamed pancreatic tissue depends on the production of chemotactic factor. The pancreatic parenchyma is actively involved in the induction of inflammation in CP through the production of different kinds of chemokines. Among other, interleukin-8, ENA78 and MCP 1 have been localized at high levels in certain tissue compartment in CP. 108 In addition, increased levels of RADTES, MIP-alpha and their receptor CCR 5, which is mainly expressed on infiltrating macrophages, have been observed in CP. 109 other factors such nerve growth factor (NGF) and receptor TrkA are expressed at increased levels in CP NGF was observed in tubular complexes and in dedifferentiating Acinar cells in CP whereas TrkA was mainly present in the perineurium in CP. 110 Interestingly, there was a strong correlation between NGF levels and pancreatic fibrosis and Acinar cell damage, as well as between TrkA levels and pain intensity. 30 It was, therefore, suggested that the NGF/TrkA pathway might influence neural morphological changes in the pain syndrome in CP.

The other major players in the transmission of pain are neurotransmitters. The same group previously demonstrated 16 that the neurotransmitters substance P is increased in the intrapancreatic nerves in CP. However, substance P function via its specific

receptor

(NK-1R), and therefore determination of the presence of NK-1R and localization of the site of NK-1R gene expression is expected to provide further insight into substance P-mediated pathways. Thus, the expression of NK-1R mRNA was quantitatively studied and the site of its mRNA and protein expression localized. We also evaluated whether there is a relationship between the presence of NK-1R mRNA levels, and pain and inflammation in CP.NK-1R was localized in the enlarged intrapancreatic nerves, ganglia blood vessels and inflammatory cells. The NK-1RmRNA levels obtained by quantitative polymerase chain reaction (PCR) were also correlated with clinical parameters. This analysis revealed that expression of the NK-1R gene correlation significantly with the intensity, duration and frequency of pain, but not with the severity of pancreatic inflammation.

ISLET CELL AUTOTRANSPLANTATION:

In one study, these patients, when treated with intrahepatic islet cell autotransplantation, did remarkably well (blood glucose <126 mg/d1, hemoglobin A1c 6.5%). Five of six patients did not require insulin therapy at all 111.The results of islet cell autotransplantation in the portal vein, within the spleen and portal vein or within the spleen alone are equally good. Even though patients require insulin, the dose needed to achieve normoglycemia is low. The embolisation technique has complication such as duodenal ischemia, splenic infarction and portal or splenic vein thrombosis. Even after surgery, over 15% of patients continue to experience pain and an end stage fibrotic gland and those dependent on narcotics do not do well after islet cell transplantation 112.

SUMMARY

We determined long term outcome and early and late morbidity and mortality, respectively in 100 patients undergoing surgery for chronic pancreatitis from 1990 through 2004 and completed atleast six months follow-up during two years period (2003 – 2004). The mean follow up was 43. 5 months. Majority of patients are tropical pancreatitis. Indications for operation were pain, (80%), suspicion of malignancy in 2%, complications involving adjacent organs (18%). Pseudocysts were present in 10% of patients. While the hospital morbidity was less (14.3 Vs 55.6%) after drainage operation than after pancreatic resections, there was no difference in hospital mortality (1.4 Vs 0 %). The best results with pain relief occurred after LR – LPJ (92.3%). The number of patients are able to work a function normally after surgery increased markedly (9 Vs 82%, P< 0.001).Late deaths occurred in four patients. Three patients had unsuspected pancreatic malignancy. One patient had developed G.I. bleed related to splenic artery pseudo aneurysm. Pancreatic insufficiency particularly diabetes mellitus improved significantly after surgery at follow-up of 3.6 years. There was significant reduction in insulin requirement and decrease in fasting and postprandial blood glucose levels. No significant improvement in exocrine insufficiency. There was no difference in changes in endocrine status between resection and drainage group.

CONCLUSION

Operative treatment of chronic pancreatitis, when indicated can be performed safely with good results in terms of pain relief, weight gain and quality of life. Significant improvement in pancreatic endocrine insufficiency can be expected. Resectional procedures associated with higher early morbidity. Unsuspected malignancy is a common cause for late deaths. Continued use of Alcohol is associated with poor relief of pain and quality of life. Various genetic factors and genetic mechanisms have been identified and these may lead to the development of novel therapeutic strategies for the management of this particularly complex disease in the near feature, thereby improving the overall outcome.

REFERENCES

1. Helmut Friess, Pascal O.Berberat, Martin Wirtz, Markus W. Buchler. Surgical treatment and long-term follow-up in chronic pancreatitis. Eur J Gastroenterol Hepatol 2002; 14:971-997.
2. J.D. Evans, P.G.Wilson, C.Carver, S.R. Bramhall, J.A.C. Bucklels, A.D.Mayer, P. McMaster, J.P. Neoptolemos. Outcome of surgery for chronic pancreatitis. British Journal of Surgery 1997; 84:624-629.
3. Thierry Berney, Tatiana Rudisuhli, Jose Oberholzer, Aileen Caulfield, Philippe Morel. Long-term metabolic results after pancreatic resection for severe chronic pancreatitis. Arch Surg. 2000; 135:1106-1111.
4. Ebbehoj N, Borly L, Madsen P, Svendsen LB. Pancreatic tissue pressure and pain in chronic pancreatitis. Pancreas 1986; 1:556-558.
5. Buchler M, Friess H, Mueller MW, et al. Randomized trial of duodenum preserving pancreatic head resection versus pylorus preserving Whipple in chronic pancreatitis. Am J Surg 1995; 169:65-70.
6. Lankisch PG, Lohr-Happe A, Otto J, et al: Natural course in chronic pancreatitis. Digestion 1993; 54:148-155.
7. Layer P, Yamamoto H, Kalthoff L, et al: The difficult courses of early- and late-onset idiopathic and alcoholic pancreatitis. Gastroenterology 1994; 107:1481.
8. Ammann RW, Akovblantz A, Largiader F,et al. Course and outcome of chronic pancreatitis. Gastroenterology. 1984; 86:820.
9. Andren-Sandberg A, Hafstrom A: Partington-Rochelle: When to drain the pancreatic duct and why. Dig Surg 1996; 13:109-112.

10. Igor Sielezneff, Andrew Malouf, Etienne Salle, Christian Brunet, Xavier Thirion, Bernard Sastre. Longterm results of lateral pancreaticojejunostomy for chronic alcoholic pancreatitis. Eur J Surg 2000; 166:58-64.

11. Adams D, Margaret C. Ford, B.A., Marion C. Anderson. Outcome after lateral pancreaticojejunostomy for chronic pancreatitis. Ann Surg 1994; 219:481-489.

12. Warshaw AL, Banks PA, Fernandez-Del Castillo C: AGA technical review: Treatment of pain in chronic pancreatitis. Gastroenterology 1998; 115:765-776.

13. Sharma AK, Pande GK, Sahni P, Nundy S: Surgery for nonalcoholic chronic pancreatitis. World J Surg 1998; 22:236-240.

14. Brinton MH, Pellegrini CA, Stein SF, Way LW: Surgical treatment of chronic pancreatitis. Am J Surg 1984; 148:754-759.

15. Jalleh RP, Williamson RC: Pancreatic exocrine and endocrine function after operations for chronic pancreatitis. Ann Surg 1992; 216:656-662.

16. Nealon WH, Townsend CM Jr, Thompson JC: Operative drainage of pancreatic duct delays functional impairment in patients with chronic pancreatitis: A prospective analysis. Ann Surg 1988; 208:321-329.

17. Sidhu SS, Nundy S, Tandon RK: The effect of the modified Puestow procedure on diabetes in patients with tropical chronic pancreatitis: A prospective study. Am J Gastroenterol 2001; 96:107-111.

18. Farkas G, Leindler L, Daroczi M, Farkas G Jr,: Organ-preserving pancreatic head resection in chronic pancreatitis. Br J Surg 2003; 90:29-32.

19. Buchler MW, Friess H, Bittner R, Roscher R. Krautzberger W, Muller MW, Malfertheiner P, Beger HG: Duodenum-preserving pancreatic head resection: Long-term results. J Gastrointest Surg 1997; 1:13-19.

20. Cameron JL, Pitt HA, Yeo Cj, Lillemoe KD, Kaufman HS, Coleman J. One hundred and forty-five consecutive pancreaticoduodenectomies without mortality. Ann Surg 1993; 217:430-438.

21. Nitecki SS, Sarr MG, Colby TV, Van Heerden JA. Long-term survival after resection for ductal adenocarcinoma of the pancreas. Is it really improving? Ann Surg 1995; 221:59-66.

22. Stapleton GN, Williamson RCN. Proximal pancreaticoduodenectomy for chronic pancreatitis. Br J Surg 1996; 83:1433-1440.

23. Ho HS, Frey CF. Current approach to the surgical management of chronic pancreatitis. Gastroenterologist. 1997; 5:128-136.

24. Heise JW, Katoh M, Luthen R, Roher H-D. Long-term results following different extent of resection in chronic pancreatitis. Hepatogastroenterology 2001; 48:864-868.

25. Sakorafas GH, Sarr MG, Rowland CM, Farnell MB. Postobstructive chronic pancreatitis: results with distal resection. Arch Surg 2001; 136:643-648.

26. Rattner DW, Fernandez-del Castillo C, Warshaw AL. Pitfalls of distal pancreatectomy for relief of pain in chronic pancreatitis. Am J Surg 1996; 171:142-146.

27. Sawyer R, Frey CF. Is there still role for distal pancreatectomy in surgery for chronic pancreatitis? Am J Surg 1994;168:6-9.

28. Sakorafas GH, Farnell MB, Farley DR, Rowland CM. Sarr MG. Long-term results after surgery for chronic pancreatitis. Int J Pancreatol 2000; 27:131-142.

29. Izbicki JR, Bloechle C, Broering DC, Knoefel WT, Kuechler T, Broelsch CE. Extended drainage versus resection in surgery for chronic pancreatitis: A prospective, randomized trial comparing the longitudinal pancreaticojejunostomy combined with local pancreatic head excision with the pylorus-preserving pancreaticoduodenectomy. ANZ Surg 1998; 228:771-779.

30. Frey CF, Amikura K. Local resection of the head of the pancreas combined with longitudinal pancreaticojejunostomy in the management of patients with chronic pancreatitis. Ann Surg 1994; 220:492-504.

31. Izbicki JR, Bloechle C, Knoefel WT, Kuechler T, Binmoeller KF, Broelsch CE.

Duodenum-preserving resection of the head of the pancreas in chronic pancreatitis. A prospective randomized trial. Ann Surg 1995; 221:350-358.

32. Izbicki JR, Bloechle C, Knoefel WT, Kuechler T, Binmoeller KF, Sohendra N, Broelsch CE. Drainage versus resection in surgical therapy of chronic pancreatitis of the head of the pancreas. A randomized study. Chirurg 1997; 68:369-377.

33. Klempa I, Spatny M, Menzel J, Baca I, Nustede , tockmann F, Arnold W. Pancreatic function and quality of life after resection of the head of the pancreas in chronic pancreas in chronic pancreatitis. A prospective, randomized comparative study after duodenum preserving resection of the head of the pancreas versus Whipple's operation. Chirurg 1995; 66:350-359.

34. Jay S. Markowitz, David W. Rattner, Andrew L. Warshaw. Failure of symptomatic relief after pancreaticojejunal decompression for chronic pancreatitis – strategies for salvage. Arch Surg 1994; 129:374-380.

35. Singh M, Simsek H. Ethanol and the pancreas. Gastroenterology 1990; 98:1051-1062.

36. Buchler M, Weihe E, Friess H, et al. Changes in peptidergic innervation in chronic pancreatitis. Pancreas 1992; 7:182-192.

37. Farney AC, Najarian JS, Nakhleh RE, et al. Autotransplantation of disperses pancreatic islet tissue combined with total or near-total pancreatectomy for treatment of chronic pancreatitis. Surgery 1991;110:427-439.

38. Strum WB: Abstinence in alcoholic chronic pancreatitis: Effect on pain and outcome. J Clin Gastroenterol 1995;20:37.

39. De las Heras G, de la Pena J, Lopez-Arias M, et al: Drinking habits and pain in chronic pancreatitis. J Clin Gastroenterol 1995;20:33.

40. Brinton M.H, Pellegrini C.A., Stein S.F., Way L.W. Surgical treatment of chronic pancreatitis. Am J Surg 1984;148:754.

41. Sato T, Miyashita E, Matsuno S, Yamauchi H. The role of surgical treatment for chronic pancreatitis. Ann Surg 1986;203:226.

42. Sharma AK, Pande GK, Peush Sahni, Samiran Nundy. Surgery for nonalcoholic chronic pancreatitis. World J Surg 1998;22:236-240.

43. Thomas P.G., Augustine P, Ramesh H., Rangabashyam N. Observations and surgical management of tropical pancreatitis. World J Surg 1990;14:32-42.

44. Augustine P. A three year study of chronic pancreatitis in Kerala. In: Balakrishnan V. ed. Chronic pancreatitis in India. Trivandrum, India: Indian society of pancreatology,1987:73-79.

45. Tripathy BB, Samal KC. Chronic calcific pancreatitis with diabetes in young in Orissa. In: Balakrishnan V. ed. Chronic pancreatitis in India. Trivandrum, India: Indian society of pancreatology,1987:87-96.

46. Nealon WH, Thompson JC..Progressive loss of pancreatic function in chronic pancreatitis is delayed by main duct decompression. Ann Surg 1993;217:458-468.

47. Hammel P, Malka D, Levy P, et al. Influence of elective pancreatic surgery on the occurrence of diabetes in patients with chronic pancreatitis. Gastroenterology 1996;110:A395 (abstract).

48. Johanns W, Janssen J, Greiner L. Extracorporeal shock wave lithotripsy- A new option for treating stone associated obstructive chronic pancreatitis. Gastroenterology 1996;110: A403 (abstract).

49. Brand B, Kahl M, Sidhu S, et al. Prospective evaluation of morphology, function and quality of life following ESWL and endoscopic treatment of chronic calcific pancreatitis. Gastrointest Endosc 1999;49:A648 (abstract).

50. Agarwal G, Sikora SS, Choudhuria G, Bhatia E. Prospective study of pancreatic β-cell and exocrine function following duct decompression in tropical calcific pancreatitis. World J Surg 2002;26:171-175.

51. Ramesh H, Augustine P. surgery in tropical pancreatitis: Analysis of risk factors. Br J Surg 1992;79:544.

52. Karanjia ND, Widdison Al, Leung F, Alvarez C, Lutrin FJ, Reber HA. Compartment syndrome in experimental chronic obstructive pancreatitis. Effect of decompressing the main pancreatic duct. Br J Surg 1994;81:259-264.

53. David Malka, Pascal Hammel, Alain Sauvanet et al. Risk factors for Diabetes Mellitus in chronic pancreatitis. Gastroenterology 2000;119:1324-1332.

54. Traverso LW, Kozarek RA. Pancreaticoduodenectomy for chronic pancreatitis. Anatomic selection criteria and subsequent long-term outcome analysis. Ann Surg 1997;226:429-438.

55. Sato N, Yamaguchi K, Yokohata K, Shimizu S, Noshiro H et al. Changes in pancreatic function after pancreaticoduodenectomy. Am J Surg 1998;176:59-61.

56. Miyata M, Yamamotot T, Hamaji M, Izukura M, et al. Pancreatic endocrine functions in long-term survivors after pancreaticoduodenectomy:special reference to reversibility of insulin and glucagons secretion. World J Surg 1988;12:651-657.

57. Yamaguchi K, Yokohata K, Nakano K, et al. Which is less invasive pancreatic head resection: PD, PPPD, or DPPHR? Dig Dis Sci 2001;46:282-288.

58. Ohtsuka T, Yamaguchi K, Chijiiwa K, et al,. Postoperative pancreatic exocrine function influences body weight maintenance after pylorus preserving pancreaticoduodenectomy. Am J Surg 2001;182:524-529.

59. DiMagno EP, Clain JE, Layer P. Chronic pancreatitis. In: Go VL, ed. The pancreas: biology, pathobiology and disease. New York: Raven Press, 1993:665-706.

60. Nealon WH, Walser E. Duct drainage alone is sufficient in the operative management of pancreatic pseudocyst in patients with chronic pancreatitis. Ann Surg 2003; 237: 614-620.

61. Vitas GJ, Sarr MG: Selected management of pancreatic pseudocysts: Operative versus expectant management. Surgery 1992;111:123-130.

62. Cheruvu CV, Clarke MG, Prentice M, EyreBrook IA: Conservative treatment as an option in the management of pancreatic pseudocyst. Ann R Coll Surg Engl 2003;85:313-316.

63. Usatoff V, Brancatisano R, Williamson RC: Operative treatment of pseudocysts in patients with chronic pancreatitis. Br J Surg 2000;87:1494-1499.

64. Nealon WH, Walser F. Main pancreatic duct anatomy can direct choice of modality for treating pancreatic pseudocysts. Ann Surg 2002;235:751-758.

65. Ng B, Murray B, Hingston G, et al. An audit of pancreatic pseudocysts management and the role of endoscopic pancreatography. ANZ J Surg 1998;68:847-851.

66. Heider R, Galanko J, Meyer AA, Behrns KE. Percutaneous drainage of pancreatic pseudocysts is associated with a higher failure rate than surgical treatment in unselected patients. Ann Surg 1999;229:781-789.

67. Beckingham IJ, Krige JE, Bornman PC, Terblanche J. Long-term outcome of endoscopic drainage of pancreatic pseudocysts. Am J Gastroenterol 1999;94:71-74.

68. Joseph D. Vijungco, Richard A. Prinz. Management of biliary and duodenal complications of chronic pancreatitis. World J Surg 2003;27:1258-1270.

69. Wapnick S, Hadas N, Purow E, et al. Mass in the head of the pancreas in cholestatic jaundice. Carcinoma or pancreatitis? Ann Surg 1979;190:587-591.

70. Frey C, Suzuki M, Isaji S. Treatment of chronic pancreatitis complicated by obstruction of the common bile duct or duodenum. World J Surg 1990;14:59-69.

71. Vitale GC, Reed DN Jr, Nguyen Ct, Lawhon JC, Larson GM. Endoscopic treatment of distal bile duct stricture from chronic pancreatitis. Surg Endosc 2000;14:227-231.

72. Kahl S, Zimmermann S, Genz I, Glasbrenner B, Pross M, Schulz HU, et al. long-term results of endoscopic treatment of biliary strictures in chronic pancreatitis: A prospective follow-up study. Am J Gastroenterol 2003;98:2448-2453.

73. Cunha JE, Machado M, Bacchella T, Penteado S, Mott CB, Jukemura J, Pinotti HW: Surgical treatment of pancreatic ascites and pancreatic pleural effusions. Hepatogastroenterology 1995;42:748-751.

74. Parekh D, Segal I. Pancreatic ascites and effusions. Risk factors for failure of conservative therapy and the role of octreotide. Arch Surg 1992;127:707-712.

75. Kozarek RA. Endoscopic therapy of complete and partial pancreatic duct disruptions. Gastrointest Endosc Clin N Am 1998;8:39-53.

76. Bernades P, Baetz A, Levy P, Belghiti J, et al. Splenic and portal venous obstruction in chronic pancreatitis. Dig Dis Sci 1992;37:340-346.

77. Knoefel WT, Luth M, Langwieler TE, Peiper M, Hosch SB, Izbicki JR Jr. Is portal vein obstruction with cavernomatous transformation of the portal vein a contraindication for surgical therapy of chronic pancreatitis? Pancreas 2000;21:456-459.

78. Lowenfels AB, Maisonneuve P, Cavallini G, Ammann RW, Lankisch PG, Andersen JR, Dimagno EP, Andren-Sandberg A, Domellof L. Pancreatitis and the risk of pancreatic cancer. International pancreatitis study group. N Engl J Med 1993;328:1433-1437.

79. Malka D, Hammel P, Maire F, et al. Risk of pancreaticadenocarcinoma in chronic pancreatitis. Gut 2002;51:849-852.

80. Lowenfels AB, Maisonneuve P, DiMagno EP, Elistur Y, Gates LK, et al. International hereditary pancreatitis and the risk of pancreatic cancer. J Natl Cancer Inst 1997;89:442-446.

81. Augustine P, Ramesh H. Is tropical pancreatitis premalignant? Am J Gastroenterol 1992;87:1005-1008.

82. Saez A, Catala I, Brossa R, Funes A, Jaurrieta E, Ferrer JE. Introperative fine needle aspiration cytology of pancreatic lesions. A study of 90 cases. Acta Cytol 1995;39:485-488.

83. Rumstadt B, Forssmann K, Singer MV, Trede M. The Whipple partial duodenopancreatectomy for the treatment of chronic pancreatitis. Hepatogastroenterology 1997;44:1554-1559.

84. Ammann RW, Muellhaupt B. Zurich Pancreatitis study group. The natural history of pain in alcoholic chronic pancreatitis. Gastroenterology 1999;116:1132-1140.

85. Dumonceau J-M, Deviere J, Le Moine O, Delhaye M, Vandermeeren A, Baize M, Van Gansbeke D, Cremer M. Endoscopic pancreatic drainage in chronic pancreatitis associated with ducatl stones: long-term results. Gastorintest Endosc 1996;43:547-555.

86. Delhaye M, Vandermeeren A, Baize, Cremer M. Extracorporeal shock-wave lithotripsy of pancreatic calculi. Gastroenterology 1992;102:610-620.

87. Smits ME, Rauws AJ, Tygat NJ, Hulbregtse K. Endoscopic treatment of pancreatic stones in patients with chronic pancreatitis. Gastrointest Endosc 1996;43:556-560.

88. Adamek HE, Jakobs R, Buttmann A, Adamek MU, Schneider ARJ, Riemann JF. Long-term follow up of patients with chronic pancreatitis and pancreatic stones treated with extracorporeal shock wave lithotripsy. Gut 1999;45:402-406.

89. Kozarek RA, Patterson DJ, Ball TJ, Traverso LW. Endoscopic placement of pancreatic stents and drains in the management of pancreatitis. Ann Surg 1989;209:261-266.

90. Dite P, Ruzicka M, Zbori V, Novotny I. A prospective, randomized trial comparing endoscopic and surgical therapy for chronic pancreatitis. Endoscopy 2003;35:553-558.

91. Gress F, Schmitt C, Sherman S, et al. Endoscopic ultrasound-guided celiac plexus block for managing abdominal pain associated with chronic pancreatitis: A prospective single center experience. Am J Gastroenterol 2001;96:409-416.

92. Gress F, Schmitt C, Sherman S, et al. A prospective,randomized comparison of endoscopic ultrasound and computed tomography-guided celiac plexus block for managing chronic pancreatitis pain. Am J Gastroenetrol 1999;94:900-905.

93. Makarewicz W, Stefaniak T, Kossakowska M, et al. Quality of life improvement after videothoracoscopic splanchnicectomy in chronic pancreatitis patients. Case control study. World J Surg 2003;27:906-911.

94. Bradley EL 3rd, Reynhout JA, Peer GL. Thoracoscopic splanchnicectomy for “small duct” chronic pancreatitis. Case selection by differential epidural analgesia. J Gastrotintest Surg 1998;2:88-94.

95. Howard TJ, Swofford JB, Wagner DL, et al. Quality of life after bilateral thoracoscopic splanchnicectomy. Long-term evaluation in patients with chronic pancreatitis. J Gastrointest Surg 2002;6:845-852.

96. Ihse I, Zoucas E, Gyllstedt E, Lillo-Gil R, Andren-Sandberg A. Bilateral thoracoscopic splanchnicectomy: Effects on pancreatic function. Ann Surg 1999;230:785-790.

97. Buchler HC, Jansen JB, Van Dongen R, et al. Long-term results of bilateral thoracoscopic splanchnicectomy in patients with chronic pancreatitis. Br J Surg 2002;89:158-162.

98. Simon P, Weiss FU, Zimmer KP et al. Spontaneous and sporadic trypsinogen mutation in idiopathic pancreatitis. JAMA 2002;288:2122.

99. Venter JC, Adams MD, Myers EW et al. The sequence of the human genome. Science 2001;291:1304-1351.

100. Friess H, Ding J, Kleff J Liao Q, Beberat PO, Hammer J, et al. Identification of disease-specific genes in chronic pancreatitis using DNA array technology. Ann Surg 2001;234:769-778.

101. Wsposito I, Friess H, Kappeler A, Shrikhande S, et al. Mast cell distribution and activation in chronic pancreatitis. Hum Pathol 2001;32:1174-1183.

102. Shrikhande S, Friess H, Zimmerman A, Ramesh H, Buechler MW. Comparison of alcoholic,idiopathic and tropical chronic pancreatitis on a molecular basis. In: Buechler MW, Friess H, Uhl W, Malfertheiner P (eds). Chronic pancreatitis: Novel concepts in biology and therapy. Berlin:Blackwell;2002:66-72.

103. Friess H, Shrikhande S, Shrikhande M Martignoni M, Kulli C, Zimmermann A, et al. Neural alterations in surgical stage chronic pancreatitis are independent of the underlying etiology. Gut 2002;50:682-686.

104. Shrikhande SV, Martignoni ME, Shrikhande M, Kappeler A, Ramesh H, Zimmermann A, et al. Comparison of histological features and inflammatory cell reaction in alcoholic, idiopathic and tropical chronic pancreatitis. Br J Surg 2003;90:1565-1572.

105. Chandak GR, Idris MM, Reddy DN, Bhaskar S, Sriram PV, Singh L. Mutations in the pancreatic secretory trypsin inhibitor gene (PSTI/SPINK 1) rather than the cationic trypsinogen gene (PRSS1) are significantly associated with tropical calcific pancreatitis. J Med Genet 2002;39:341-351.

106. Shrikhande S, Friess H, Buechler M. Pathogenesis of pain in chronic pancreatitis. In: Wig JD (ed). The pancreas. Chandigarh: Azad publishers;2000:352-357.

107. Di Sebastiano P, Fink T, Weihe E, Friess, Innocenti P, Beger HG, et al. Immune cell infiltration and growth-associated protein 43 expression correlate with pain in chronic pancreatitis. Gastroenterology 1997;112:1648-1655.

108. Saurer L, Reber P, Schaffner T, et al. Differential expression of chemokines in normal pancreas and in chronic pancreatitis. Gastroenterology 2000;118:356-367.

109. Goecke H, Forssman U, Uguccioni M et al. Macrophages infiltrating the tissue in chronic pancreatitis express the chemokines receptor CCR 5. Surgery 2000;128:806-814.

110. FriessH, Zhu ZW, di Mola FF et al. Nerve growth factor and its high-affinity receptor in chronic pancreatitis. Ann Surg 1999;230:615-624.

111. Robertson RP, Lanz KJ, Sutherland DE, Kendall Dm. Prevention of diabetes for up to 13 years by autoislet transplanatation after pancreatectomy for chronic pancreatitis. Diabetes 2001;50:47-50.

112. White SA, Davies JE, Pollard C, et al. Pancreas resection and islet autotransplantation for end-stage chronic pancreatitis. Ann Surg 2001; 233:423-31.

Parameters	Case1	Case2	Case 3	Case 4	Case 5	Case 6	Case7	Case 8
Age/sex	57/M	25/F	28/F	38/F	21/M	45/M	54/M	53/M
Etiology	Alcohol	Tropical	Pancreas divisum	Tropical	Tropical	Alcohol	Alcohol	Alcohol
Symptom Duration	6 months	12 years	4 months	3 months	1 year	7 yrs	2 mon	3 yrs
Indication For surgery	Pseudocyst	Pain	Pain	Jaundice	Pseudocyst	Pain	? Ca. Head	GI Bleed
Steatorrhea	No	No	No	No	No	No	Yes	No
Analgesic use	Injectable	Injectable	Oral	Injectable	Oral	Oral	Oral	Oral

Diabetes	Nil	Nil	Nil	Nil	Nil	Nil	Yes	Yes
Insulin (U)	-	-	-	-	-	-	20	No
OHA							-	Yes
Blood sugar (F/PP)							138/316	198/411
Morphology								
MPD	8 mm	1 cm	7 mm	3 cm	1.1 cm	6 mm	7 mm	4 mm
Ductal calculi	+	+	+	+	+	+	+	-
Parenchymal	+	+	-	-	+	-	-	-
Calcification								
Others	-	-	Pan. divisum	-	-	-	Bulky head	Cyst in head 6x3 cm Blood clots+
Associated Complications	Pseudocyst	Abscess Tail	GSD	CBD stricture	Pseudocyst	Abscess Body & Tail	-	GDA Pseudo Aneurysm
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ +Cysto-jejunostomy	DP + PJ	LPJ + Chole	LPJ + CDD	LPJ	DP	PD	Excision of Pseudoaneur-ysm with PJ
Postoperative Complications	Wound infection	Nil	Nil	Nil	Nil	Nil	Nil	Pancreatic Fistula – Conser. Mx
Follow-up								
Pain relief	Fair	Fair	Good	Good	Good	Good	Good	Good
Weight gain	4 Kg+	3 Kg+	4 Kg+	4 Kg+	5 Kg+	4 Kg+	2 Kg+	4 Kg+
Steatorrhea	Occasional	No	No	No	No	No	Yes	No
Req. for enzymes	No	No	No	No	No	No	Yes	No
Req. for Insulin (U)	20	50	No	No	No	No	No	No
Req. for OHA	No	No	No	No	No	Yes	Yes	No
GTT/ Bl. sugar (F/PP)	Control	Control	Normal	Normal	Normal	Control	126/214	112/136
Alcohol withdrawal	Yes	NA	NA	NA	NA	Yes	Yes	Yes
Employment status	No	No	No	Yes	Yes	Yes	Yes	Yes
Rehospitalisation	1-Pain Abd (2/2k)	No	No	Pain abd.	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	7 years	7 years	5 years	7 years	5 yrs	6 yrs	8 yrs	1 1/2 Yrs

Parameters	Case 9	Case 10	Case 11	Case 12	Case 13	Case 14	Case 15	Case 16
Age/sex	40/M	64/M	52/M	39/M	48/F	49/M	57/M	32/F
Etiology	Alcohol	Tropical	Alcohol	Alcohol	Tropical	Alcohol	Hereditary	Tropical
Symptom Duration	1 month	1 year	1 month	5 yrs	1 year	1 mo	1 mon	1 mo
Indication For surgery	Jaundice	Pain	Pain	Jaundice	Pseudocyst	Jaundice	Jaundice	Pain
Steatorrhea	No	No	No	No	Yes	No	Yes	No
Analgesic use	Injectable	Injectable	Injectable	oral	Oral	Oral	Oral	Injectable
Diabetes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Insulin (U)	No	No	No	35	No	No	45	45
OHA	Yes	Yes	No	No	Yes	Yes	No	No
Blood Sugar (F/PP)	73/228	144/276	124/192	272/360	140/250	225/310	190/356	188/280
Morphology								
MPD	3 cm	1 cm	1 cm	7 mm	7 mm	8 mm	1 cm	2.5 cm
Ductal calculi	+	+	+	-	+	+	+	-
Parenchymal Calcification	+	+	-	-	+	+	-	-
Others	Mass Neck Atrophied Pancreas	Atrophied Pancreas	-	Bulky head 4.7 cm	-	Atrophy	-	-
Associated Complications	CBD Stricture	Abscess Body& Tail	GSD	CBD stricture	Abscess Body	CBD Stricture	CBD Stricture	-
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ +CDD	DP + LPJ	PG + Chole	LPJ + CDD	LPJ + Ext. drainage	LPJ + CDD	LPJ + CDD	LPJ
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Follow-up								
Pain relief	Poor	Good	Good	Good	Fair	Fair	Fair	Fair
Weight gain	20 Kg loss	3 Kg+	4 Kg+	7 Kg+	9 Kg+	7 Kg+	3 Kg+	Same
Steatorrhea	No	No	No	No	Yes	No	No	No
Req. for enzymes	No	No	No	No	Yes	No	No	No
Diabetic status	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Req. for Insulin(U)	No	No	No	No	No	No	No	10
Req. for OHA	No	Yes	No	Yes	Yes	Yes	Yes	No
GTT/ Blood sugar	86/126	128/189	106/148	162/242	164/244	146/234	156/234	148/282
Alcohol withdrawal	Yes	NA	NA	Yes	NA	Yes	NA	NA
Employment status	No	No	No	Yes	No	Yes	Yes	Yes
Rehospitalisation	2-Vomiting &Pain Abd	No	No	No.	No	No	No	No
Reoperation	1-Dissem.Ca.head GOO - GJ done	No	No	No	No	No	No	No
Overall assessment	Poor	Good	Good	Good	Good	Good	Good	Good
Outcome	Died	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	$1_{1/2}$ yrs	5 years	5 years	8 years	9 yrs	8 yrs	11 yrs	13 Yrs

Parameters	Case 17	Case 18	Case 19	Case 20	Case 21	Case 22	Case 23	Case 24
Age/sex	44/F	30/F	28/F	25/M	32/M	19/M	28/M	30/M
Etiology	Tropical	Tropical	Tropical	Tropical	Alcohol	Hereditary	Tropical	Tropical
Symptom Duration	18 mo	1 year	1 Yr	2 yrs	3 mon	3 yrs	5 year	4 mo
Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pain	Pain	Pain
Steatorrhea	No	No	No	No	No	No	Yes	No

Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Oral	Injectable
Diabetes	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Insulin (U)	No	40	55	45	No	54	45	12
OHA	Yes	No	No	No	Yes	No	No	No
Blood sugar (F/PP)	85/180	330/458	246/348	276/412	208/352	248/382	170/295	192/332
Morphology								
MPD	1 cm	9 mm	1 cm	1 cm	1 cm	9 mm	8 mm	1.5 cm
Ductal calculi	+	+	+	+	+	+	+	+-
Parenchymal Calcification	-	-	-	-	+	+	+	+
Others	-	Pseudocyst Head 2x3 cm	-	PD Stricture At Body	-	Uncinate stones	Atrophy	-
Associated Complications	Nil	Ileocaecal Mass	Nil	Nil	Nil	Nil	Nil	Nil
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ	LPJ + Enteroent-ostomy	LPJ	LPJ	LPJ	LPJ	Puestow's	LPJ
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Follow-up								
Pain relief	Good	Fair	Fair	Good	Good	Good	Good	Good
Weight gain	4 Kg+	5 Kg+	5 Kg+	7 Kg+	5 Kg+	same	3 Kg+	3 Kg+
Steatorrhea	No	No	No	No	No	No	No	No
Req. for enzymes	No	No	No	No	Yes	No	No	No
Diabetic status	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Req. for Insulin(U)	No	No	No	20	No	37	10	No
Req. for OHA	No	Yes	Yes	No	Yes	No	No	Yes
GTT/ Blood sugar	112/156	169/302	158/248	146/262	164/224	196/294	230/298	136/264
Alcohol withdrawal	NA	NA	NA	NA	NA	Yes	NA	NA
Employment status	No	No	No	Yes	Yes	Yes	Yes	Yes
Rehospitalisation	1-Pain Abd	No	1-Abd. Pain	No.	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	14 yrs	15 yrs	4 years	15 Yrs	14 yrs	12 yrs	12 yrs	4 Yrs

Parameters	Case 25	Case 26	Case 27	Case 28	Case 29	Case 30	Case 31	Case 32
Age/sex	27/M	27/M	35/F	35/M	22/M	33/F	42/M	48/M
Etiology	Tropical	Tropical	Tropical	Alcohol	Tropical	Tropical	Alcohol	Alcohol
Symptom Duration	1 mo	2 yrs	3 mo	8 mo	2 ½ Yrs	5 mo	1 mo	15 yrs

Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pain	Pain	Pain
Steatorrhea	No	No	No	No	No	No	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Insulin (U)	12	12	No	No	8	30	16	45
OHA	No	No	Yes	Yes	No	No	No	No
Blood sugar (F/PP)	266/348	198/340	180/312	154/299	124/220	242/320	222/334	240/396
Morphology								
MPD	1 cm	7 mm	1 cm	1 cm	1 cm	6 mm	1.5 cm	8 mm
Ductal calculi	+ Head	+	+	+	+	+	+	+
Parenchymal Calcification	-	-	-	+	+	- Atrophy	+	+
Others	Atrophy	-	-	-	-	Pus in PD	Head mass	Pus in PD
Associated Complications	Nil	Nil	Nil	Nil	Nil	Miliary Abd. TB	Nil	Nil
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Follow-up								
Pain relief	Good	Good	Good	Good	Good	Good	Good	Good
Weight gain	12 Kg+	12 Kg+	3 Kg+	5 Kg+	5 Kg+	3 Kg+	7 Kg+	5 Kg+
Steatorrhea	No	Occasional	No	No	No	No	No	No
Req. for enzymes	No	No	No	No	Yes	No	No	No
Diabetic status	No	Yes	No	Yes	Yes	Yes	Yes	Yes
Req. for Insulin(U)	No	12	No	No	8	No	No	No
Req. for OHA	No	No	No	Yes	No	Yes	Yes	Yes
GTT/ Blood sugar	100/346	154/272	104/136	172/264	134/214	128/236	140/274	148/252
Alcohol withdrawal	NA	NA	NA	Yes	NA	NA	Yes	Yes
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rehospitalisation	Nil	No	No	No.	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	3 yrs	3 yrs	3 years	1 Yr	3 yrs	3 yrs	4 yrs	2 Yrs

Parameters	Case 33	Case 34	Case 35	Case 36	Case 37	Case 38	Case 39	Case 40
Age/sex	40/M	51/M	34/M	32/F	59/M	28/F	55/F	31/F
Etiology	Alcohol	Alcohol	Tropical	Tropical	Tropical	Tropical	Tropical	Tropical
Symptom	3 yrs	1 ½ yrs.	7 yrs	10 yrs	3 mo	6 mo	3 yrs	2 yrs

Duration								
Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pain	Pain	Pain
Steatorrhea	No	Yes	No	No	No	No	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
Insulin(U)	No	46	45	No	30	No	45	No
OHA	Yes	No	No	Yes	No	No	No	Yes
Blood sugar (F/PP)	134/258	188/332	205/376	196/278	248/372	-	236/398	194/346
Morphology								
MPD	1.5 cm	1 cm	1.2 cm	8 mm	9 mm	1 cm	6 mm	1 cm
Ductal calculi	+ Head	+	+	+	+	+	+	+
Parenchymal Calcification	+	+	+	+	+	+	-	+
Others	Atrophy	Atrophy Head mass	Atrophy Head mass	Atrophy Head mass	Head mass ? Ca.	Atrophy	-	-
Associated Complications	Nil	Pseudocyst head	Nil	Nil	Nil	Nil	Nil	Pseudocyst tail
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	Frey's	Frey's	Frey's	Frey's	Frey's	LPJ	LPJ	LPJ
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Atelectasis	Wound infection
Follow-up								
Pain relief	Good	Good	Good	Good	Good	Good	Good	Good
Weight gain	3 Kg+	4 Kg+	4 Kg+	same	2 Kg+	3 Kg+	3 Kg+	3 Kg+
Steatorrhea	No	Occasional	No	No	No	No	No	No
Req. for enzymes	No	No	No	No	Yes	No	No	No
Diabetic status	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
Req. for Insulin	No	12	10	12	No	No	No	No
Req. for OHA	Yes	No	No	No	Yes	No	Yes	Yes
GTT/ Blood sugar	128/238	152/246	135/226	168/284	164/278	-	174/246	146/258
Alcohol withdrawal	Yes	Yes	NA	NA	NA	NA	NA	NA
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rehospitalisation	Nil	No	No	No.	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Good	Poor	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Death	Alive	Alive	Alive
Follow-up period	1 yr.	1 yr	1 year	1 Yr	10 months Ca.head	4 yrs	5 yrs	1 Yr

Parameters	Case 41	Case 42	Case 43	Case 44	Case 45	Case 46	Case 47	Case 48

Age/sex	30/M	21/F	30/M	26/F	43/F	50/M	24/M	35/M
Etiology	Tropical	Tropical	Tropical	Tropical	Tropical	Alcohol	Tropical	Tropical
Symptom Duration	2 yrs	4 yrs.	1 yr	1 mo	12 yrs	3 mo	4 yrs	10 yrs
Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pain	Pain	Pain
Steatorrhea	Yes	Yes	No	No	No	Yes	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Insulin(U)	18	56	21	20	30	45	No	No
OHA	No	No	No	No	No	No	Yes	Yes
Blood Sugar (F/PP)	229/378	304/412	228/338	222/278	282/384	312/482	105/220	176/288
Morphology								
MPD	1 cm	8 mm	1 cm	7 mm	4 mm	6 mm	1 cm	1.5 cm
Ductal calculi	+ Head	+	+	-	+	+	+	+
Parenchymal Calcification	+	+	+	+	+	+	-	+
Others	-	Atrophy	Atrophy	-	Atrophy Head mass	Atrophy	Pus in PD	-
Associated Complications	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ	LPJ	LPJ	LPJ	Frey's	LPJ	Puestow's	LPJ
Postoperative Complications	Nil	Atelectasis	Nil	Wound infection	Nil	Wound infection	Nil	Wound infection
Follow-up								
Pain relief	Good	Good	Good	Good	Good	Poor	Fair	Good
Weight gain	3 Kg+	2 Kg+	9 Kg+	3 kg loss	4 Kg+	2 Kg loss	6 Kg+	4 Kg+
Steatorrhea	No	Occasional	No	No	No	No	No	No
Req. for enzymes	No	No	No	Yes	No	No	No	No
Diabetic status	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Req. for Insulin(U)	No	20	No	No	No	No	No	No
Req. for OHA	Yes	No	Yes	Yes	Yes	Yes	No	Yes
GTT/ Blood sugar	138/272	186/248	166/234	158/256	148/238	182/268	98/124	136/218
Alcohol withdrawal	NA	NA	NA	NA	NA	Yes	NA	NA
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rehospitalisation	Nil	No	No	No.	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Fair	Good	Fair	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	1 yr.	1 yr	2 yrs	1 1/2 Yr	3 yrs	1 1/2 yrs	12 yrs	3 Yrs

Parameters	Case 49	Case 50	Case 51	Case 52	Case 53	Case 54	Case 55	Case 56
Age/sex	52/M	33/M	18/F	34/M	35/M	32/M	40/M	43/M
Etiology	Alcohol	Tropical	Tropical	Alcohol	Tropical	Tropical	Tropical	Alcohol
Symptom Duration	1 yrs	1 yr.	2 yrs	2 mo	1 mo	3 yrs	3 mo	5 yrs
Indication	Pain	Pain	Pain	Pain	Pain	Pain	Pain	?

For surgery								Malignancy
Steatorrhea	No	Yes	No	No	Yes	Yes	No	Yes
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Oral
Diabetes	Yes	Yes	Yes	Yes	Yes	Yes	No	No
Insulin (U)	18	12	No	No	No	12	-	-
OHA	No	No	Yes	Yes	Yes	No	-	-
Blood sugar (F/PP)	246/312	205/325	236/318	144/296	198/316	118/200	-	-
Morphology								
MPD	1.2 cm	1 cm	1 cm	8 mm	5 mm	9 mm	3 cm	4 mm
Ductal calculi	+	+	+	+	+	+	+	-
Parenchymal Calcification	+	+	-	-	+	+	-	+
Others	-	Atrophy	Strictures at Body & Tail	-	Atrophy Head mass	Atrophy	Pus in PD	Head mass 6x5 cm Atrophy-
Associated Complications	Pseudocyst head	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ	LPJ	LPJ	LPJ	Frey's	LPJ	LPJ	Whipple's
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Gastropare-sis	Bile leak operated
Follow-up								
Pain relief	Good	Good	Good	Good	Good	Fair	Poor	Good
Weight gain	3 Kg+	21 Kg loss	4 Kg+	4 Kg+	5 Kg+	5 Kg+	4 Kg loss	1 Kg loss
Steatorrhea	No	Yes	No	No	Yes	Yes	No	Yes
Req. for enzymes	No	Yes	No	No	Yes	Yes	No	Yes
Diabetic status	No	No	Yes	Yes	Yes	Yes	No	No
Req. for Insulin (U)	No	No	No	No	No	No	No	No
Req. for OHA	No	No	Yes	Yes	Yes	Yes	No	No
GTT/ Blood sugar	96/126	96/128	136/224	146/244	142/278	128/216	Normal	Normal
Alcohol withdrawal	Yes	NA	NA	Yes	NA	NA	NA	Yes
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Rehospitalisation	Nil	No	No	No	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Fair	Good	Good	Good	Good	Poor	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Dead	Alive
Follow-up period	1 yr.	3 yrs	1 yr	8 Yrs	3 yrs	3 yrs	1 yrs Ca.head	1 Yr

Parameters	Case 57	Case 58	Case 59	Case 60	Case 61	Case 62	Case 63	Case 64
Age/sex	25/M	55/M	55/M	12/M	24/F	13/M	38/M	22/F
Etiology	Tropical	Alcohol	Tropical	Tropical	Tropical	Tropical	Alcohol	Tropical

Symptom Duration	7 yrs	10 mo	5 yrs	2 yrs	4 mo	3 yrs	3 Yrs	2 yrs
Indication For surgery	Pain	Pain	Pain	Pain	Pancreatic Ascites	Pancreatic Ascites	Pancreatic Ascites	Pain
Steatorrhea	No	No	No	No	Yes	Yes	Yes	Yes
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	No	No	No	No	No	No	No	No
Insulin	-	-	-	-	-	-	-	-
OHA	-	-	-	-	-	-	-	-
Morphology								
MPD	1 cm	2 cm	4 mm	9 mm	8 mm	7 mm	7 mm	4 mm
Ductal calculi	+	+	+	+	+	+	+	+
Parenchymal Calcification	+	+	+	-	+ Head	+	+	+
Others	Head mass	Head mass	Head mass Necrosis+	Head mass	Ductal dis-ruptions	Ductal dis-ruptions	Duct disru-ption Tail Stricture Neck	Head mass
Associated Complications	Nil	Nil	Nil	Nil	Ascites	Ascites	Ascites	Pseudocyst head
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	Frey's	Frey's	Frey's	Frey's	PG	PG	DP + PJ	Frey's
Postoperative Complications	Nil	Nil	Pancreatic Leak	Nil	Nil	Nil	Pancreatic leak	Nil
Follow-up								
Pain relief	Good	Good	Good	Good	Good	Good	Good	Good
Weight gain	4 Kg+	4 Kg+	8 Kg+	4 Kg+	3 Kg+	4 Kg+	3 Kg loss	2 Kg+
Steatorrhea	No	No	No	No	No	No	No	No
Req. for enzymes	No	Yes	No	No	No	No	No	Yes
Diabetic status	No	No	No	No	No	No	No	No
Req. for Insulin	No	No	No	No	No	No	No	No
Req. for OHA	No	No	No	No	No	No	No	No
GTT/ Blood sugar	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Alcohol withdrawal	NA	NA	NA	NA	NA	NA	Yes	NA
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Rehospitalisation	Nil	No	No	No	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	6 mo	1 1/2 yrs	2 yrs	1 Yr	7 mo	1 yr	9 mo	6 mo

Parameters	Case 65	Case 66	Case 67	Case 68	Case 69	Case 70	Case 71	Case 72
Age/sex	43/M	31/M	28/M	31/F	25/M	18/M	22/M	36/M
Etiology	Alcohol	Tropical	Tropical	Tropical	Tropical	Tropical	tropical	Alcohol
Symptom Duration	1 yr	11 yrs	20 yrs	2 yrs	3 mo	4 yrs	5 Yrs	6 mo

Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pain	Pain	Pain
Steatorrhea	No	No	No	No	No	No	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	No	No	No	No	No	No	No	No
Insulin	-	-	-	-	-	-	-	-
OHA	-	-	-	-	-	-	-	-
Morphology								
MPD	1 cm	1 cm	2.5 cm	7 mm	7 mm	1.4 cm	7 mm	7 mm
Ductal calculi	+	+	+	+	+	+	+	+
Parenchymal	+	+	+	-	+ Head	+	+	+
Calcification								
Others	Head mass Pus in PD	Atrophy Stent in PD In situ	Pus in PD	-	Stricture Body	Atrophy	-	Atrophy
Associated Complications	Nil	Nil	Nil	Nil	Nil	GB sludge	Nil	Nil
Interventions								
Endoscopic	Nil	PD stent 6 mo	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	Frey's	PG	LPJ	LPJ	LPJ	LPJ + Chole	LPJ	LPJ
Postoperative Complications	UTI	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Follow-up								
Pain relief	Poor	Good	Poor	Good	Good	Good	Good	Good
Weight gain	5 Kg loss	5 Kg+	4 Kg+	6 Kg+	4 Kg+	3 Kg+	4 Kg+	3 Kg+
Steatorrhea	Yes	No	No	No	No	No	No	No
Req. for enzymes	Yes	Yes	No	No	No	No	No	No
Diabetic status	No	No	No	Yes	No	No	No	No
Req. for Insulin	No	No	No	No	No	No	No	No
Req. for OHA	No	No	No	(diet control)	No	No	No	No
GTT/ Blood sugar	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Alcohol withdrawal	Yes	NA	NA	NA	NA	NA	Yes	Yes
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Rehospitalisation	1-Pain abd	No	2-Pain abd.	No	No	No	No	1-Pain abd
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Poor	Good	Fair	Good	Good	Good	Good	Fair
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	2 yrs	3 yrs	3 yrs	2 1/2 Yrs	3 yrs	1 yr	1 ½ yrs	1 yr

Parameters	Case 73	Case 74	Case 75	Case 76	Case 77	Case 78	Case 79	Case 80
Name	Anbarasan	Vasanthi	Karpagam	Anbarasi	Arulkumar	Bhabya	Venkata	Lavanya
Age/sex	33/M	49/F	32/F	19/F	24/M	23/F	65/M	20/F

Etiology	Tropical	Tropical	Tropical	Tropical	Tropical	Tropical	tropical	Tropical
Symptom Duration	1 mo	3 yrs	1 mo	4 yrs	2 mo	10 yrs	30 Yrs	1 yr
Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pain	Pain	Pain
Steatorrhea	No	No	No	No	No	No	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	No	No	No	No	No	No	No	No
Insulin	-	-	-	-	-	-	-	-
OHA	-	-	-	-	-	-	-	-
Morphology								
MPD	2 cm	8 mm	7 mm	9 mm	8 mm	8 mm	1.5 cm	6 mm
Ductal calculi	+	+	+	+	+	+	+	+
Parenchymal Calcification	+	-	+	+	+	+	+	+
Others	-	-	Strictures at Head+	-	-	-	-	-
Associated Complications	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Interventions		Pancre. Sphin-						
Endoscopic	Nil	Cterotomy+	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Follow-up								
Pain relief	Good	Fair	Good	Good	Good	Good	Good	Good
Weight gain	4 Kg+	same	4 Kg+	3 Kg+	7 Kg+	3 Kg+	same	4 Kg+
Steatorrhea	No	No	No	No	No	No	No	No
Req. for enzymes	No	No	No	No	No	No	No	No
Diabetic status	No	No	No	No	No	No	No	No
Req. for Insulin	No	No	No	No	No	No	No	No
Req. for OHA	No	No	No	No	No	No	No	No
GTT/ Blood sugar	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Alcohol withdrawal	NA	NA	NA	NA	NA	NA	Yes	No
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Rehospitalisation	No	No	No	No	No	No	No	No
Reoperation	Nil	No	No	No	No	No	No	No
Overall assessment	Good	Good	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	1 yr	11/2 yr	1 ½ yrs	1 1/2 Yrs	1 yr	3 yrs	10 mo	10 mo

Parameters	Case 81	Case 82	Case 83	Case 84	Case 85	Case 86
Age/sex	42/M	15/F	18/M	16/M	37/M	37/M
Etiology	Tropical	Tropical	Tropical	Tropical	Alcohol	Alcohol
Symptom Duration	10 yrs	1 ½ yrs	6 mo	2 mo	2 yrs	1 yrs
Indication for surgery	Pain	Biliary stenosis	Pain	GI Bleed	Pain	Pain
Steatorrhea	occasional	Yes	No	No	No	No
Analgesic use	Injectable	Oral	Injectable	Oral	Injectable	Injectable
Diabetes	No	No	No	No	No	Yes
Insulin (U)	-	-	-	-	-	45
OHA	-	-	-	-	-	-
Blood sugar (F/PP)	-	-	-	-	-	318/456

Morphology						
MPD	1 cm	6 mm	8 mm	8 mm	4 mm	1 cm
Ductal calculi	+	+	+	-	-	+
Parenchymal	+	-	-	-	+	+
Calcification				-	Multiple	
Others	-	Atrophy	Atrophy		strictures	PD Stent+
Associated Complications	Nil	CBD stricture	Nil	SA Pseudo-aneurysm bleed	Pseudocyst Head 3x2.5cm	Nil
Interventions						
Endoscopic	Nil	Nil	Nil	Nil	Nil	PD Stent
Surgical	LPJ	LPJ + CDD	LPJ	Excision of Pseudoaneu-rysm with Central Pancreatectomy With PJ	Frey's	LPJ
Postoperative Complications	Nil	Nil	Nil	Nil	Wound Dehiscence GI Bleed Operated	Nil
Follow-up						
Pain relief	Good	Good	Good	Good	-	Poor
Weight gain	3 Kg+	4 Kg+	3 Kg+	4 Kg+	-	6 Kg loss
Steatorrhea	No	No	No	No	--	Yes
Req. for enzymes	No	No	No	No	-	Yes
Diabetic status	No	No	No	No	-	Yes
Req. for Insulin(U)	No	No	No	No	-	20
Req. for OHA	No	No	No	No	-	No
GTT/ Blood sugar	Normal	Normal	Normal	Normal	-	244/310
Alcohol withdrawal	NA	NA	NA	NA	-	No
Employment status	Yes	Yes	Yes	Yes	-	No
Rehospitalisation	No	No	No	No	-	Yes
Reoperation	Nil	No	No	No	Reoperated Pancreatic bleed MODS	Ruptured Splenic Artery PseudoAneurysm DP +Excision of PA Gastric perforation
Overall assessment	Good	Good	Good	Good		Closure +Transverse
Outcome	Alive	Alive	Alive	Alive		Colostomy
Follow-up period	7 mo	2 yrs	3 yrs	3 Yrs	Death	Death – 6 months
Parameters	Case 87	Case 88	Case 89	Case 90	Case 91	Case 92
Age/sex	31/M	43/M	43/M	36/M	12/F	43/M
Etiology	Alcohol	Tropical	Alcoholic	Alcoholic	Tropical	Tropical
Symptom Duration	2 Yrs	2 mo	6 mo	6 mo	2 yrs	2 yrs
Indication for surgery	Pain	Pain	Pain	Pain	GI Bleed	Haemoptysis & GI Bleed
Steatorrhea	No	No	No	No	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Oral
Diabetes	Yes	Yes	Yes	Yes	No	No
Insulin (U)	No	35	30	12	-	-
OHA	Yes	-	No	No	-	-

Blood sugar(F/PP)	76/96	346/416	378/476	195/350	-	-
Morphology						
MPD	1 cm	1 cm	6 mm	8 mm	5 mm	1 cm
Ductal calculi	+	+	+	+	-	+
Parenchymal	+	+	+	+	+	
Calcification				+	-	
Others	-	Pus in PD Atrophy	-	Atrophy		-
Associated Complications	Nil	Resolving Pseudocyst Lt. Subhepatic	Nil	Nil	Splenic artery Pancreatic duct Communication Ruptured pseudocyst Intraperitoneal bleed	Pseudoaneurysm of Splenic Artery eroding into Diaphragm, bronchus and colon
Interventions						
Endoscopic	Nil	Nil	Nil	Nil	Nil	ESWL + stenting done
Surgical	LPJ	LPJ	LPJ	LPJ	DP+Roux-n-PJ	DP+ Excision of SA Pseudoaneurysm+ Closure of diaphragmatic And colonic rent
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil
Follow-up						
Pain relief	Good	Good	Good	Good	Good	Good
Weight gain	3 Kg+	4 Kg+	5 Kg+	4 Kg+	2 Kg+	3 Kg+
Steatorrhea	No	No	No	No	No	No
Req. for enzymes	No	No	No	No	No	No
Diabetic status	Yes	Yes	Yes	Yes		
Req. for Insulin	No	No	No	No	No	No
Req. for OHA	Yes	Yes	Yes	Yes	No	No
GTT/ Blood sugar	116/178	248/306	236/318	146/212	Normal	Normal
Alcohol withdrawal	Yes	No	Yes	Yes	NA	NA
Employment status	No	Yes	No	Yes	Yes	Yes
Rehospitalisation	No	No	No	No	No	No
Reoperation	No	No	No	No	No	Colostomy closure
Overall assessment	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	7 mo	1 ½ Yrs	2 Yrs	2 ½ Yrs	6 months	1 yr.

Parameters	Case 93	Case 94	Case 95	Case 96	Case 97	Case 98	Case 99	Case 100
Age/sex	52/M	49/M	55/M	35/M	46/F	66/M	23/F	31/M
Etiology	Tropical	Alcohol	Alcohol	Alcohol	Tropical	Tropical	Tropical	Alcohol
Symptom Duration	3 mo	2 mo	3 yrs	1 yr	2 mo	4 mo	1 Yr	5 yrs
Indication For surgery	Pain	Pain	Pain	Pain	Pain	Pseudocyst	Pain	GI Bleed
Steatorrhea	No	No	No	No	No	No	No	No
Analgesic use	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable	Injectable
Diabetes	Yes	Yes	No	No	No	No	No	No
Insulin (U)	No	8	-	-	-	-	-	-
OHA	Yes	No	-	-	-	-	-	-
Blood sugar (F/PP)	135/234	136/324						

Morphology								
MPD	7 mm	1.2 cm	8 mm	9 mm	9 mm	1 cm	1.5 cm	6 mm
Ductal calculi	+	+	+	+	+	+	+	-
Parenchymal	-	+	+	+	-	-	-	-
Calcification						-		
Others	-	-	Atrophied	Mass Tail & Body	Atrophy	-	-	
Associated Complications	Nil	Nil	Nil	Nil	Nil	Pseudocyst Head + GSD	Gallstone	Splenic Artery Pseudoaneurysm Pseudocyst Body
Interventions								
Endoscopic	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil
Surgical	LPJ	LPJ	LPJ	LPJ	LPJ	LPJ +Cysto-Gastrostomy+ Chole	LPJ+ Chole	Transcystic Ligation of Bleeding Vessel Cystogastrostomy
Postoperative Complications	Nil	Nil	Nil	Nil	Nil	Nil	Pancreatic leak	Nil
Follow-up								
Pain relief	Good	Good	Good	Good	Good	Good	Good	Good
Weight gain	3 Kg+	4 Kg+	4 Kg+	4 Kg+	3 Kg+	4 Kg+	4 Kg+	3 Kg+
Steatorrhea	No	No	No	No	No	No	No	No
Req. for enzymes	No	No	No	No	No	No	No	No
Diabetic status	Yes	Yes	No	No	No	No	No	No
Req. for Insulin(U)	No	No	No	No	No	No	No	No
Req. for OHA	Yes	No	No	No	No	No	No	No
GTT/ Blood sugar	116/229	118/168	Normal	Normal	Normal	Normal	Normal	Normal
Alcohol withdrawal	NA	NA	NA	Yes	NA	NA	Yes	No
Employment status	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Rehospitalisation	No	No	No	No	No	1 - Incisional Hernia Repair	No	No
Reoperation	Nil	No	No	No	No		No	No
Overall assessment	Good	Good	Good	Good	Good	Good	Good	Good
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Follow-up period	4 yrs	3 yrs	7 yrs	3 Yrs	3 yrs	5 yrs	5 yrs	2 yrs